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THE OSTEOGENETIC GRAFT IN TUBERCULOSIS OF THE HIP-JOINT IN CHILDHOOD

BY E. D. TELFORD
EMERITUS PROFESSOR OF SURGERY, UNIVERSITY OF MANCHESTER

AND J. E. GEDDES
MEDICAL SUPERINTENDENT, THE MANCHESTER SANATORIUM, ABERGELE

THE ideal result of treatment of a tuberculous joint is recovery with free and painless movement, but in view of the fact that in the fight against the disease the components of the joint are transformed into granulation tissue, the complete recovery of the joint is not likely to be attained by any form of treatment. The odd case which shows complete recovery is, no doubt, an error in diagnosis. Such joints as recover with fibrous ankylosis and some movement may be labelled for the time as recoveries, but experience shows that they are prone to give much trouble in later life from relapse, vicious position, and osteo-arthritic changes.

In the present state of our knowledge the best result is a solid bony fusion in the optimum position, and this result is not often obtained by conservative treatment alone. For these reasons the bone-graft has been extensively used in the treatment of tuberculous disease of the hip-joint. The technique is varied and often ingenious, but the methods may be divided into two groups: (1) The fixative graft; (2) The osteogenetic graft.

1. THE FIXATIVE GRAFT

This graft, which is no more than an internal splint, is usually extra-articular in position. It is liable to fracture or to fail in union at one or other end, and the great variety of methods employed suggests that the results are not entirely satisfactory.

2. THE OSTEOGENETIC GRAFT

A review of the literature of the past ten years shows that this graft has been used less frequently than the fixative graft, possibly owing to the fear of opening a diseased joint. In our experience this fear is without foundation.

The osteogenetic graft, properly employed, should be used only in a joint already quiescent and on its way to repair. It is in no sense fixative, but acts purely as a stimulus to osteogenesis. Some writers think that its action is due to the implant of calcium, and maintain that other substances, e.g., chalk, will produce a like result. We prefer to think that the osteogenesis is due to the action of fresh osteoblasts acting on a granulation tissue which is stagnant and unable of itself to make bone. It is perhaps permissible to hope that in the future it may be possible to ask the tissue-culture laboratory to provide osteoblasts for injection into the joint and so do away with the need for open operation.

If an early case of tuberculosis of the hip-joint in a child is placed under adequate fixation it will be found that after a period of destruction, sometimes widespread and rapid, there occur signs of arrest and consolidation at the end of 18 to 20 months. If the conservative treatment is maintained, few of these joints will end in bony ankylosis, but if, when definite radiological evidence of arrest and repair is seen, a graft is inserted into the joint, the majority of these hip-joints will develop a rapid and diffuse osteogenesis which goes on to complete bony fusion.

The Operation.—With the child lying on its sound side, an incision 5 to 6 in. long is made in the line of the femur, the prominence of the trochanter marking the mid-point of the incision. This incision is carried down to bone throughout its length. The upper edge of the acetabulum is identified, and immediately below it a gouge is entered into the remains of the joint space, and a narrow but deep track is made in the softened bone and granulation tissue.

The graft is taken from the trochanter and adjacent part of the femoral shaft. This graft is 2½ to 3 in. in length and ⅓ to ½ in. in width. The periosteum is stripped off the required area and the graft cut by an osteotome or electric saw. The graft consists, as to one half, of trochanter;

the remaining half is the compactum of the femoral shaft. The graft is completely detached, and the femoral end is pushed into the cavity which has been made in the joint; the trochanteric end will be found to fit accurately into the bed from which it was cut, and is fixed by a catgut suture. The divided muscles are sutured and the wound closed without drainage.

The limb is placed in the optimum position of slight flexion and abduction, and a double plaster spica is applied.

This spica is renewed to allow clinical and radiological examination every three months, and in the average case the child is allowed up in a short plaster or celluloid spica at the end of a year. The average length of post-operative treatment before discharge free of any appliance is a little over two years.

Valuable evidence as to prognosis is given by the state of the graft as seen in serial radiographs. Rapid loss of outline and disappearance of the graft indicates the likelihood of a favourable result, but persistence of a well-defined graft after 9 to 12 months foreshadows a delayed or incomplete result.

Indications.—The most important of these is that the operation be not done until radiological evidence of arrested disease and commencing repair is seen.

The operation should not be done in very young children. In two cases in which we operated at 6 years and 7 years there was failure to obtain firm fusion. Eight years, or, possibly better, 10 years, should be the earliest.

The presence of closed abscess is no contra-indication—these may exist and even increase side by side with clear evidence of repair, and such abscesses commonly yield to one aspiration, or, if their contents are thick, to expression through a small incision with immediate suture. These procedures would be carried out sometime before the operation on the hip.

We have not operated in the presence of an active sinus, but have done so in several cases where a sinus has been well healed for three months.

Some evidence of tuberculous infection in the radiograph of the chest is, of course, a commonplace in these children, but any evidence of recent activity in the chest would be a contra-indication.

Results.—Between 1932 and 1940 we have done this operation on 26 children whose ages ranged from 6 years to 18 years. Fifteen of these patients were in the North Wales Sanatorium at Llangwyfan, and we are indebted to Dr. F. S. Hawkins for very valuable help in a recent follow-up of these cases.

There was no mortality or morbidity in the series—there would appear to be little risk of either local implantation or general dissemination after open interference with these joints. All the 26 cases have been traced and their present condition is known; it has been possible to bring up 20 of them for clinical and radiological examination. Nineteen are well and are in active work or at school. The operated hips are solidly fused, free from ache or pain, and show no deterioration in position.

The remaining 7 are all well and active, and although they might be claimed as 'recoveries', the presence of a few degrees of movement, usually in flexion, shows that the operation has failed to secure a bony fusion.

In reviewing the causes of these failures it seems clear that in 2 instances the patients were too young, 6 years and 7 years, whilst in 4 others our view of the radiological evidence of repair would appear in the light of further experience to have been too optimistic. In the remaining child, the existence of a pulmonary focus which re-activated at intervals no doubt supplies the explanation.

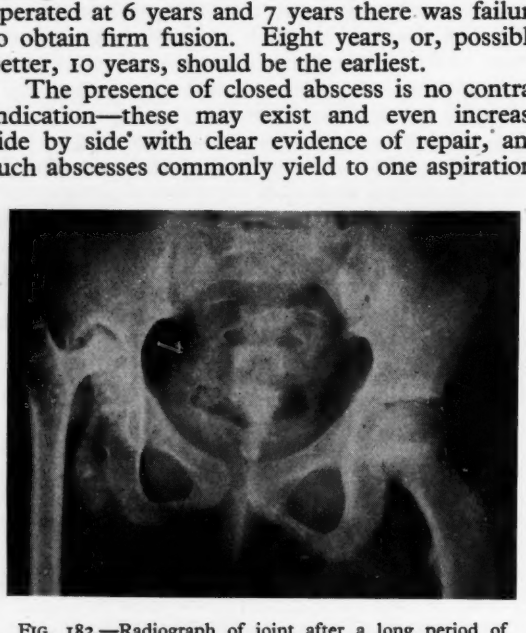

FIG. 182.—Radiograph of joint after a long period of fixation, showing failure of immobilization.

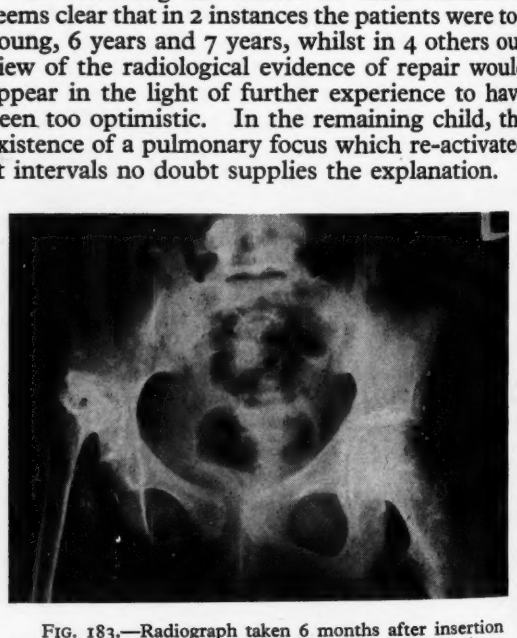

FIG. 183.—Radiograph taken 6 months after insertion of osteogenetic graft, showing bone formation throughout joint and disappearance of outline of graft.

Short notes of a typical example of successful bony fusion are as follows:—

Girl, aged 8½ years, had suffered from tuberculous disease of the right hip-joint for 3½ years before admission. After a lengthy period of fixation it was evident that there was no improvement in the joint,

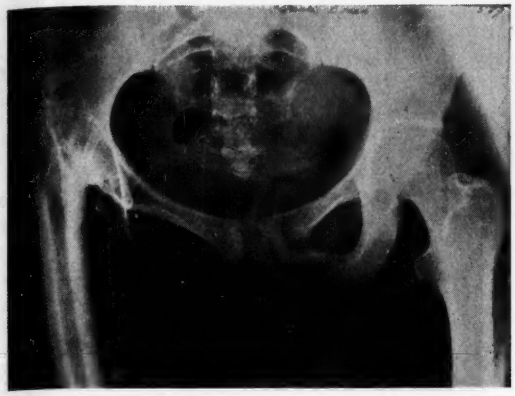

FIG. 184.—Radiograph taken 7 years after operation, showing firm fusion in good position.

the condition of which is shown in *Fig.* 182. A graft was inserted in September, 1935, and the operation was followed by rapid osteogenesis. The film (*Fig.* 183), taken six months after the operation, shows formation of bone throughout the joint—the outline of the graft is largely lost. Complete fixation was, in fact, found on clinical examination six months after operation. When seen in June, 1942, the hip was found firmly fused in good position (*Fig.* 184). Her general condition was excellent and the girl was in active work.

SUMMARY

1. The rationale and technique of the osteogenetic graft are discussed.

2. Indications and results are described.

3. Out of 26 cases, 19 gave excellent results; the remaining 7 failed to secure a firm bony fusion.

We are indebted to Dr. D. P. Sutherland, Tuberculosis Officer for Manchester, for permission to report the Abergele cases, and for much help in tracing the patients.

HYDRONEPHROSIS OF A PELVIC ECTOPIC KIDNEY

BY ROBERT H. F. BRAIN

SURGEON, E.M.S.

THE following case we think is sufficiently interesting to merit publication.

CASE REPORT

HISTORY.—A. S., male, aged 34, was admitted to hospital on Dec. 28, 1941, complaining of lower abdominal pain and swelling of from 7 to 8 years' duration.

The attacks are of an intermittent nature, occurring on an average once or twice a year. Each consists of hypogastric pain of dull aching character, followed 24 hours later by a swelling which may take up to 4 to 5 days to develop fully, although the whole thing has been known to come up overnight. A steady retrogression then follows, the whole cycle taking from 7 to 10 days. Associated with this, is a decrease in urine output during the first stage, and a corresponding increase during the second. His present attack began two days prior to admission to hospital. The taking of excessive quantities of beer has been known to bring on such an attack. No other micturition or bowel symptoms. General health excellent. Previous history and family history have nothing relevant.

ON EXAMINATION.—On admission a well-nourished and healthy man, co-operative.

Abdomen: Revealed a smooth circular swelling of about 6½ in. in diameter, situated with its centre just to the right of the midline in the hypogastrium. The edges were well defined, and it was cystic in consistency. Mobility was limited, more so from side to side than from above down. It could be lifted up from the pelvic brim. On palpation it appeared to be only just deep to the anterior abdominal wall and was uniformly dull to percussion, also just palpable by rectal examination.

C.N.S: Normal.

Chest: Evidence of slight bronchitis.

Temperature, pulse, and respiration rate were all normal.

At this stage a fairly confident diagnosis of a urachal cyst was made, and investigations were set afoot in an effort to prove this.

SPECIAL INVESTIGATIONS.—

Urine: On admission a trace of albumin and an occasional pus cell only.

Cystoscopy: Showed what appeared to be a small ureterocele in the region of the right ureteric orifice; the latter, however, appeared to be closed, i.e., no efflux came from it despite what might be described as 'hyperperistalsis' of the whole swelling. An effort to catheterize the ureter at this time failed owing to a defective instrument.

Cystoscopy was repeated two days later, when it was found that there was now a definite efflux from the right ureter, and that the surrounding swelling had disappeared; 50 c.c. of a 10 per cent bromide solution was then injected and radiographs taken.

Radiography:

1. *Straight plate* showed nothing of importance.

2. *Retrograde pyelography* showed (*Fig.* 185): (*a*) The catheter had proceeded for about ½ in. only on its usual course before describing a small circle and running abruptly to the region of the left sacro-iliac joint. (*b*) The abdominal swelling now stood out fairly distinctly as a circular shadow.

3. *Excretion pyelography* demonstrated the normal size, shape, and position of the left kidney, with no evidence of a right one.

Renal Function Tests: These were all excellent.

Urine: When examined while the swelling was retrogressing showed numerous pus cells.

PROGRESS.—The abdominal swelling rapidly disappeared and a measured intake and output of fluids showed an excess output of 35 oz. during the two days taken for it to disappear completely. Afterwards, by clinical examination a mass could just be detected on

palpation, about 2½ in. in diameter, in the region of the right sacro-iliac joint.

DIAGNOSIS.—This could now be made—namely, intermittent hydronephrosis occurring in a pelvic ectopic kidney.

Fig. 185.—Retrograde pyelogram showing the course of the catheter and the shadow of the abdominal swelling.

TREATMENT.—An operative removal of the kidney was advised in view of the risk of developing a pyonephrosis and/or other complications.

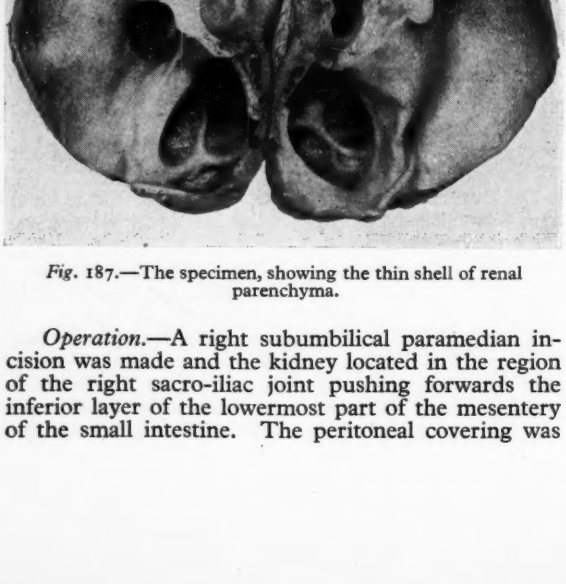

Fig. 187.—The specimen, showing the thin shell of renal parenchyma.

Operation.—A right subumbilical paramedian incision was made and the kidney located in the region of the right sacro-iliac joint pushing forwards the inferior layer of the lowermost part of the mesentery of the small intestine. The peritoneal covering was incised, and the kidney was found to be lying with its long axis at right angles to the right common iliac vessels, with the pelvis looking directly forwards and the ureter coursing over its surface in the form of a loop, convex to the left. The kidney was dissected

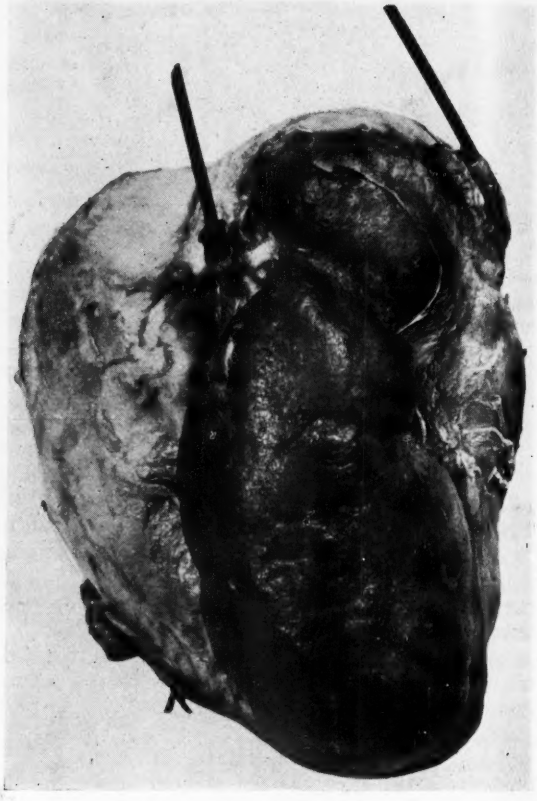

Fig. 186.—The kidney after removal (*see* text).

free and was then found to have two separate and distinct pedicles (*Fig.* 186), arising, one on the medial side of the upper pole and running to the lower end of the abdominal aorta, and the other on the lateral side of the upper pole running from there downwards and medially to join the common iliac vessels. The ureter, which appeared quite undilated, was removed down as far as the bladder wall.

POST-OPERATIVE PROGRESS.—This was quite uneventful.

THE SPECIMEN.—Examination of the specimen revealed that the renal vessels ran around the cortex, one on each side, to hila situated at the junction with the pelvis. A very large hydronephrosis was present, with only a thin shell of actual renal parenchyma remaining (*Fig.* 187).

DISCUSSION

1. The case here reported illustrates a comparatively rare condition of an ectopic kidney in the upper part of the pelvis.

2. It promotes an interesting differential diagnostic discussion, especially when considered as the case originally presented itself with a cystic abdomino-pelvic tumour.

3. The presence during the first cystoscopic examination of an extremely interesting intravesical swelling giving the appearance of a ureterocele, and the post-operative examination of the specimen, failed to reveal any evidence of dilatation of the lower end of the ureter. Explanation of this, I imagine, is due to reflex stimulation of the ureter in response to the dilated renal pelvis producing a state of 'hyperperistalsis' and a swelling due to muscle spasm.

4. The specimen on dissection well illustrated some of the developmental features of kidneys and their migrating blood-supplies, commencing at the internal iliacs and ending by obtaining their blood from the inferior suprarenal artery. Thus the specimen under consideration had obviously undergone a partial ascent to a position where its blood-supply was a dual one from both the common iliac vessels and the abdominal aorta.

5. Pathologically, the case illustrates the liability of ectopic kidneys to such conditions—in this instance a very large hydronephrosis, due apparently to a mechanical obstruction, perhaps with added mild infection and œdema at the uretero-pelvic junction.

6. Lastly, it emphasizes the importance of a full investigation of all such cases as this before attempting an operative cure. Otherwise, it may happen that the operator would find himself in the position of discovering a congenital abnormality of one kidney without the full knowledge of the position, function, etc., of the other. This, no doubt, is especially true when one is dealing with cystic conditions in the female pelvis, where perhaps operative procedures may occasionally be undertaken rather lightheartedly when dealing with the commonplace.

CARCINOMA OF THE LUNG: THE VALUE OF SPUTUM EXAMINATION IN DIAGNOSIS

By F. J. SAMBROOK GOWAR

FROM THE DEPARTMENT OF THORACIC SURGERY, THE LONDON HOSPITAL

THE surgical treatment of carcinoma of the lung has made rapid strides in the last few years, and there is to-day a reasonable hope of achieving a radical cure of the disease if the diagnosis is made sufficiently early. At the present time diagnosis is made, in the majority of cases, when the growth is well advanced, and only about 10 per cent of cases are potentially operable when first diagnosed; of these not more than 60 per cent are likely to be found operable when exploratory thoracotomy is undertaken (Tudor Edwards, 1938). Operability depends on a number of factors—site of growth, non-involvement of important structures, absence of metastases, absence of gross inflammatory complications such as suppurative pneumonitis or empyema, and the general condition of the patient. The earlier the diagnosis can be made, the more cases will be found operable. Any method, therefore, which may lead to an earlier diagnosis, or which may enable a suspicion of neoplasm to be confirmed, will enable the surgeon to find more cases suitable for operation.

The presence of carcinoma cells in the sputum has always been regarded as a rarity. Beale (1860) described masses of malignant cells in the sputum from a case of advanced pharyngeal carcinoma. Betschart (1895) described 4 cases of malignant disease of the lung in which pieces of tumour were expectorated. Weller (1913), in an analysis of 90 cases of primary bronchial carcinoma, stated that malignant cells had been found in the sputum from 4 cases. The same writer, in 1929, stated: "the pathologist may on rare occasions make a diagnosis from fragments of the tumour discovered in the sputum". Brunn (1926) stated: "the sputum should be examined thoroughly as several cases of carcinoma of the lung have been diagnosed by the finding of tumour shreds in it. These may appear rather early in the case, especially when there are intrabronchial growths". According to Fishberg (1926) the sputum may occasionally contain malignant cells, while Zemansky (1928) was of the opinion that "intra-pulmonary growths, unless originating in a bronchus, do not yield particles in the sputum". He found only 2 positive sputa in 80 cases of pulmonary cancer. Tuttle and Womack (1934), speaking of sputum-section, said, "occasionally necrotic masses in which the skeleton of the cells could be seen, were found. This was generally in those patients who were in a far advanced stage of the disease, and little valuable information was obtained. The rapid autolysis of the tumour cells in purulent sputum probably renders this method of examination of little practical value". The majority of these workers examined the sputum in the form of stained sections.

Dudgeon and Wrigley (1935) published a detailed account of a method for the examination of the sputum for evidence of malignant growth. These authors used the wet-film method which was introduced for the study of new growths and of normal and pathological tissues by Dudgeon and Patrick (1927), and investigated in greater detail by Dudgeon and Barrett (1934). Further papers by Dudgeon (1936) and Barrett (1939) showed that in a large proportion of cases of primary and secondary growth of the lung, particles of growth could be demonstrated in the sputum. Dudgeon reported that out of 56 cases

of proved or probable growth of the lung or respiratory tract, in 43 cases (i.e., 76·8 per cent) the sputum was positive. The special advantage of this method over sputum-section is that likely portions of the sputum can be selected for examination.

In spite of these striking results the method does not appear to have found favour outside St. Thomas's Hospital. McGibbon and Baker-Bates (1941), reviewing a series of cases subjected to bronchoscopy (including 263 cases of proved

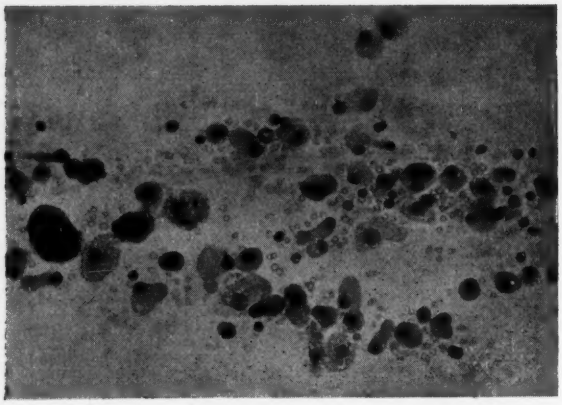

FIG. 188.—Sputum smear showing alveolar phagocytes (dust cells), and red and white blood-corpuscles. (× 212.)

or probable carcinoma), stated: "In our series, in spite of routine examination of all patients' sputum, only in a few advanced cases of malignant bronchial neoplasm have we received positive reports as to the presence of malignant cells or tumour masses."

In 1937–8, while First Assistant in the Department of Thoracic Surgery at the London Hospital, I adopted Dudgeon's method to examine the sputum from all cases of suspected pulmonary cancer referred to the Thoracic and Pathological Departments. As the technique is described in detail in Dudgeon's original papers, it will only be outlined briefly here.

Technique of Sputum Examination.—The sputum should be expectorated first thing in the morning if possible, so that there is no contamination with food particles. Instructions must be given that the specimen is to be coughed up, else the patient is frequently content merely with clearing his throat, with the result that the specimen consists only of saliva. Preservatives or disinfectants must not be added, and the specimen should be examined as soon as possible after collection. Specimens collected at bronchoscopy, or coughed up shortly afterwards, are also very suitable for examination. A certain amount of autolysis occurs if the specimen is kept, and alteration of cell structure increases the difficulty of making a positive diagnosis; however, in spite of this I was able in some cases to confirm Dudgeon's statement that "in samples sent long distances through the post, particles of malignant growth could be demonstrated, and the structure of the individual cells was preserved so well that the type of growth could be determined without difficulty."

Dudgeon advised that the sputum be poured on to an unglazed porcelain tile to concentrate it, but I found that by examining it in a large Petri dish on a black background it was easier to pick out portions for examination. Blood-streaked fragments if possible, or the more solid portions, are picked out with a platinum loop or scalpel and spread fairly thinly on a slide. Several slides (e.g., 6) are prepared from each specimen of sputum. They are fixed by immersing at once in a bath of Schaudinn's solution and leaving for 20 minutes, washing in 70 per cent alcohol containing a trace of iodine to remove excess mercuric chloride (2 min.), and then in distilled water. The slides are stained with Mayer's hæmalum (1½ to 2 min.), blued in tap water, counter-stained with eosin (2 min.), dehyrated, and mounted in Canada balsam. They are then systematically examined with the low power of the microscope, and any suspicious group of cells is submitted to a higher magnification. In cases where the clinical evidence is strongly in favour of carcinoma, several specimens of sputum should be examined before a negative report is made. A negative report does not exclude malignant disease, but has only the same value as a negative report for tubercle bacilli in a suspected case of pulmonary tuberculosis.

Histological Appearances.—Sputum normally contains a variety of cells, of which the chief are squamous epithelial cells from the upper respiratory tract and mouth, and alveolar cells containing a variable quantity of dust particles from the lung (*Fig.* 188). In pathological conditions of the lung other cells appear—pus-cells, lymphocytes, eosinophils—and, if there is destruction of lung tissue, e.g., in lung abscess, clumps of swollen alveolar cells may appear. Occasionally small clumps of columnar epithelial cells from the bronchial wall are found, especially after bronchoscopy. These clumps of cells have to be distinguished from malignant cells, and until a fair degree of experience is obtained, give rise to diagnostic difficulties. Malignant cells occur in clumps of varying size—they stain more deeply than other portions of the sputum and are picked out fairly easily under the low power of the microscope. A diagnosis of malignancy cannot be made on the appearance of a solitary cell, although this may arouse suspicion. Malignant cells are frequently found lying in a streak of blood. The principal varieties of malignant cells encountered are:—

1. *Oat-cell Carcinoma.*—The individual cells have very little cytoplasm and occur in small or large clumps. The nuclei vary in size and shape and stain deeply blue; they are large and finely granular with a well-defined nucleolus (*Fig.* 189). The cells are easily distinguished from lymphocytes, which are smaller, regular, less basophil, and do not occur in clumps.

2. *Squamous-cell Carcinoma.*—The cells are large and irregular in size and shape. Cytoplasm is abundant and stains deeply with eosin, making detection of the cells easy under the low power of the microscope (*Fig.* 190). Nuclei vary in size and shape and contain one or more well-defined nucleoli; as a rule, some of the nuclei are hyperchromatic. Multinuclear giant-cells may be found, and sometimes there is definite keratinization. With this type of growth there is a tendency for the cells to occur singly or in small clumps. The malignant cells have to be distinguished from: (*a*) Swollen alveolar cells which may occur in small clumps (*Fig.* 191); they have smaller, regular, and evenly stained nuclei which are not hyperchromatic, although cells with double nuclei may be seen; phagocytosis of pus-cells may be observed. (*b*) Squamous epithelial cells, which are large, stain faintly with eosin, and have small pale and regular nuclei. (*c*) Bronchial epithelium which may appear in the form of a cell-clump—here again the nuclei are less deeply stained and more regular; by careful focusing of the microscope cilia may be detected under the high power (*Fig.* 192).

3. *Columnar-cell Carcinoma.*—The individual cells are large with abundant cytoplasm which may be vacuolated. Groups of these cells usually have a papilliferous appearance. The nuclei are large, irregular, and deeply basophil.

4. *Undifferentiated Carcinoma* (*Fig.* 193).—In a number of cases, clumps of cells are found with the irregular darkly-staining nuclei and hyperchromatism characteristic of malignancy, but the appearances are not typical of any of the main cell-types. These cases are classified as undifferentiated carcinomata; in the majority of them bronchoscopic biopsy has shown the growth to be an undifferentiated squamous-cell type.

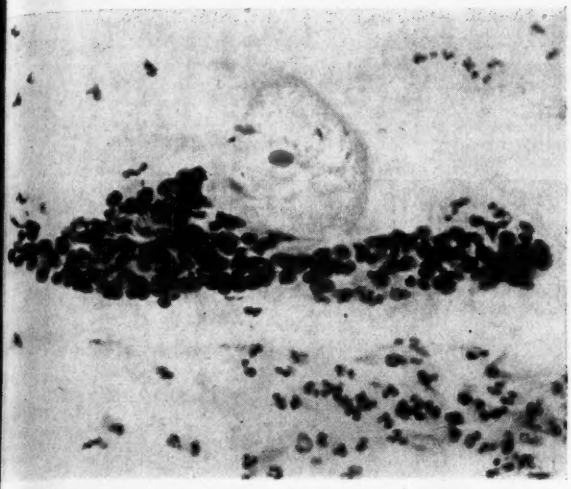

FIG. 189.—Sputum smear showing a fragment of oat-cell carcinoma; a salivary corpuscle and a number of pus-cells are also seen. Note the irregularity and hyperchromatism of the nuclei of the malignant cells. (Dufay-colour.) (× 325.)

As a rule, the cell-type of the growth diagnosed by sputum examination was found to agree with that determined by histological section, but in a few cases there was a difference. This is not surprising, for the pleomorphism of pulmonary carcinomata is well known, and an undifferentiated

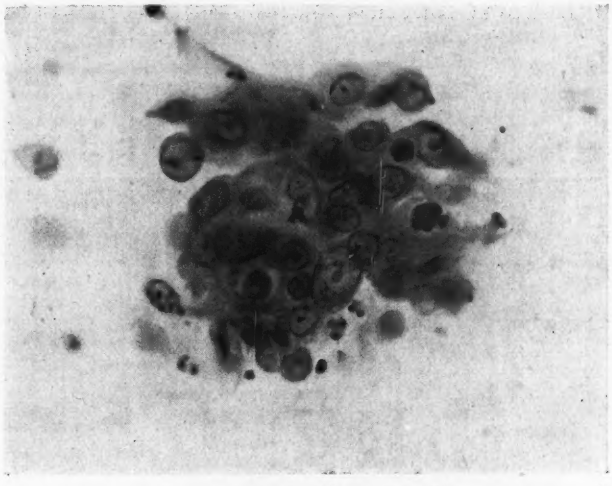

FIG. 190.—Sputum smear showing a fragment of squamous-cell carcinoma. Note the large irregular nuclei with prominent nucleoli; some of the cells are intensely eosinophil and have hyperchromatic nuclei—this appearance is characteristic of squamous-cell carcinoma. A few pus cells are also present. (Dufay-colour.) (× 400.)

FIG. 191.—Sputum smear showing a clump of swollen alveolar cells, a number of pus-cells, and part of a salivary corpuscle. From a case of diabetic gangrene of lung. (× 400.)

type of growth may produce areas resembling oat-cells, areas of squamous cells, and even areas of columnar cells.

Results.—These may be summarized as follows: Total number of cases investigated = 93. (238 specimens of sputum—an average of 2·5 per case.)

1. Cases diagnosed as new growths of lung in which fragments of growth were found in the sputum = 36.

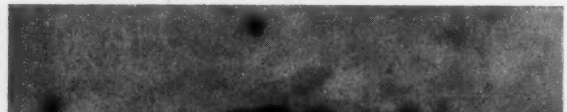

FIG. 192.—Sputum smear showing a fragment of ciliated bronchial epithelium. Fragments of squamous-cell carcinoma were also found in other parts of this smear; bronchoscopy had been performed a few days earlier and was negative. (× 440.)

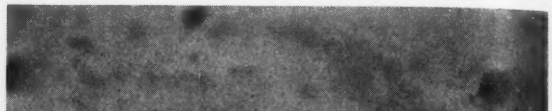

FIG. 193.—Sputum smear showing a clump of undifferentiated carcinoma cells. One cell shows a suggestion of vacuolation, and the growth was thought probably to be a columnar-cell growth. A large number of red blood-corpuscles are present. (× 392.)

2. Proved cases of new growth of lung or respiratory tract, but no growth found in the sputum = 27.

3. Suspected new growth of lung—no evidence in the sputum, and ultimately found to be non-malignant = 30.

Of the total of 93 cases, 65 were confirmed or probable primary growths of the lung, and of these 36 had a positive sputum (64·3 per cent), and 20 had a negative sputum. Of the 36 cases with a positive sputum, in 21 the diagnosis was confirmed by histological section—usually bronchoscopic biopsy—and in the remaining 15 by the subsequent course of the disease. Five cases had secondary growths in the lung or pleura, and there were 2 cases of carcinoma of the upper respiratory tract, but in none of these were fragments of growth found in the sputum. The remaining 29 cases were regarded as non-malignant on further investigation; they were chiefly inflammatory conditions of the lung, or intrathoracic tumours not arising from the lung.

Of the 36 cases with positive sputum, 13 were potentially operable—5 of these refused operative treatment and in 8 exploratory thoracotomy was undertaken; in 4 of the latter pneumonectomy was performed, while in the remaining 4 cases the growth was found to be inoperable. Of the 20 cases of primary carcinoma with negative sputum only 5 were potentially operable—2 refused operation, and in the remainder the growth was found to be inoperable at thoracotomy.

Table I.—TYPE OF GROWTH

CELL-TYPE	PROVED BY BIOPSY	NOT CONFIRMED HISTOLOGICALLY	TOTAL	PERCENTAGE
Squamous-cell	12	4	16	44·4
Oat-cell	5	2	7	19·4
Columnar-cell	0	2	2	5·6
Polygonal-cell	2	1	3	8·3
Undifferentiated or indeterminate	2	6	8	22·2
Total	21	15	36	100·0

Type of Growth.—Where the cell-type determined by sputum examination differs from that determined by histological section, the latter

is taken as being correct. The results are summarized in *Table I.*

An interesting feature of this table is the high proportion of squamous-cell growths and the low proportion of oat-cell growths. In Barrett's series of 110 primary growths of the lung examined by sputum smear, 50 per cent were of the oat-cell type, 33·6 per cent columnar, and 16·4 per cent squamous. In the majority of the published series of pulmonary growths (most of them based on post-mortem material) oat-cell types have predominated (Simpson, 1928; Maxwell, 1930; Bonser, 1934; El Gazayerli, 1936), and in a personal analysis of 110 cases which came to autopsy at the Middlesex Hospital between 1919 and 1941, I found 55·4 per cent oat-cell tumours, 16·4 per cent columnar, and 22·7 per cent squamous.* The only large post-mortem series with a predominance of squamous-cell growths is that of Atkin (1931), who found 48·8 per cent squamous-cell growths in 80 cases. Ormerod (1937) analysed 100 cases of pulmonary carcinoma diagnosed by bronchoscopic biopsy: 62 per cent were squamous, 9 per cent columnar, and 29 per cent of other types, including oat-cell types. Both Atkin's and Ormerod's series came from the Brompton Hospital. Clerf and Crawford (1933) reported a series of 50 carcinomata diagnosed by bronchoscopic biopsy, and in which also there was a high proportion of squamous-cell growths. Commenting on its greater frequency here than in post-mortem material, they suggested that it might be because squamous growths involved the larger bronchi and caused more clinical symptoms than other types. Gebauer (1941) reported 158 cases of carcinoma diagnosed histologically at biopsy or autopsy: 38·8 per cent were squamous, 33·6 per cent oat-cell, 20·2 per cent columnar, and 7·6 per cent undifferentiated. Gloyne (1937), in a paper on the cytology of sputum, commented on the greater frequency with which squamous-cell growths were detected in the sputum, and suggested that it was due to early breaking down of these growths, whereas oat-cell carcinoma does not ulcerate till a late stage and does not readily break down. Probably the discrepancies in the figures are in part accounted for by the pleomorphic nature of many of the tumours, and by the different criteria adopted by different pathologists in classifying them.

The Clinical Value of Sputum Examination.—The relative simplicity of the technique, and the fact that it causes no discomfort to the patient, together with the high proportion of cases in which a positive diagnosis can be made, give the method considerable clinical value. Contrary to the opinion held in many quarters, it is not only the advanced case which is likely to give a positive sputum test; a positive result can be anticipated in a considerable proportion of early cases. This is not surprising, for it is now generally accepted that the great majority of pulmonary carcinomata arise in the wall of a bronchus, and ulceration occurs relatively early as indicated by the occurrence of hæmoptysis in many cases. The test can be done on out-patients, and, in a suspected case occurring in general practice, the medical practitioner may send specimens of sputum to the pathologist for examination; this may be done while the patient is awaiting further investigation. It must be emphasized that a negative report does not exclude malignant disease and must not be allowed to cause postponement of further investigation in a suspected case. Close collaboration is necessary between clinician and pathologist in the collection of suitable specimens and in sending them for examination as soon as possible. At the same time the pathologist needs to spend a considerable amount of time in acquainting himself with the technique and the microscopical appearances before he can report on the smears with confidence. To the surgeon the test is valuable in some cases in clinching the diagnosis of malignancy where it has not been possible to obtain definite proof by any other means. He can thus put the case for operation more strongly to the patient and is able to proceed with greater confidence. Sweany (1934) reported 1 case in which bronchoscopy was negative, but the diagnosis was made by sputum-section. Barrett (1939) described 4 such cases in which the diagnosis was made by sputum-smear, and there were several in my series. The test is also of value in establishing the diagnosis in patients who are too ill to be submitted to bronchoscopy or other methods of investigation. Examination of the sputum for malignant cells should not, however, be regarded as an alternative to the usual methods of investigation (except in the last group of cases); it should be done in conjunction with these.

CASE REPORTS

The above points are illustrated by the following case reports.

Case 1.—**Carcinoma of Lung simulating Chronic Lung Abscess.**

W. V., male, aged 60, a road-sweeper, complaining of winter cough for 5 years. There was some shortness of breath, and 10 weeks before admission to hospital he had coughed up a little blood. Examination: A wasted old man, no fever, sputum 3 to 10 oz. daily, bloodstained. Lungs: percussion note impaired, diminished breath-sounds, and a few râles at the right base. X rays showed a thick-walled cavity at the right base (*Figs.* 194, 195), and only a small bronchial connexion could be demonstrated by bronchography. The sputum was examined, but owing to inexperience with the method at that time, a negative report was made. (Later, re-examination of the slides showed numerous clumps of squamous carcinoma cells, many of them degenerate.) A diagnosis of chronic lung abscess was made, and drainage was performed in two stages, as the pleura was found to be free at the first operation. When the abscess was opened at the second-stage operation, its wall showed a few whitish nodules, portions of

* I am indebted to Professor J. McIntosh for permission to quote these figures.

which were taken for section: the histological report was squamous-cell carcinoma. This patient improved temporarily after the operation, but died a few months later from secondary hæmorrhage.

The chief point about this case is that had the sputum been reported positive pre-operatively, an attempt might have been made to remove the lung if the patient's general condition had allowed it. The

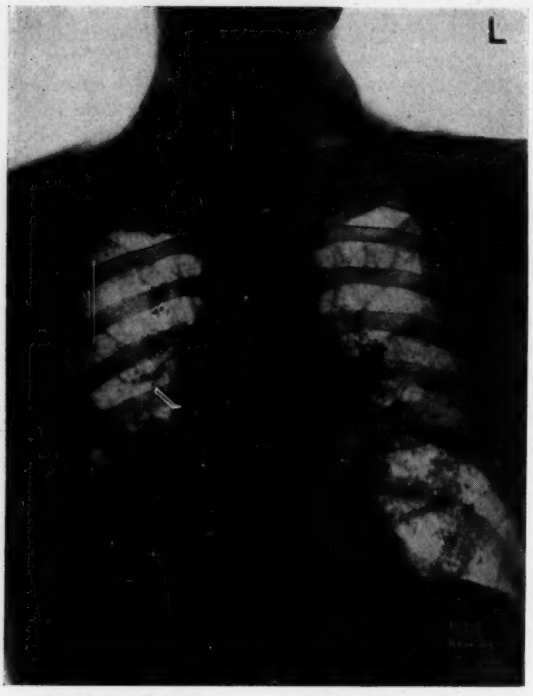

FIG. 194.—*Case* 1. Radiograph of chest showing a large thick-walled cavity with a fluid level at the right base. Much residual neohydriol present in the lungs. There is some enlargement of the right hilar shadow. This case was eventually proved to be one of squamous-cell carcinoma with cavitation.

FIG. 195.—Same case as *Fig.* 194. Lateral radiograph of chest.

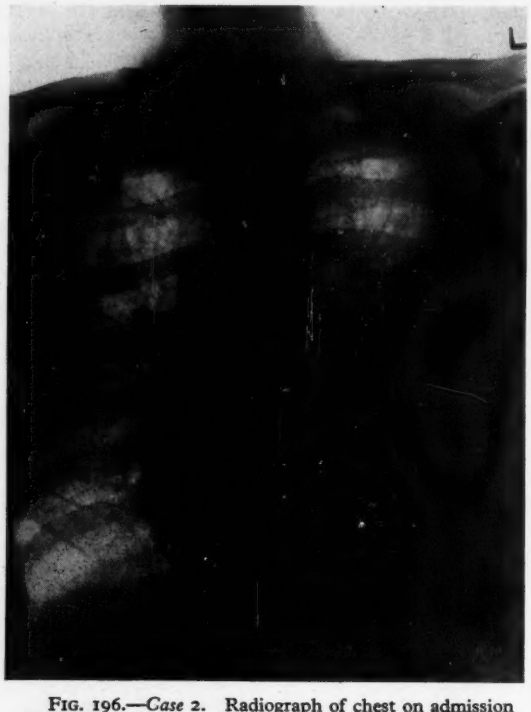

FIG. 196.—*Case* 2. Radiograph of chest on admission showing left basal empyema. The mediastinum is displaced to the right.

FIG. 197.—Same case as *Fig.* 196. After drainage of empyema: lateral bronchogram showing 'rat-tail' obstruction of left lower lobe bronchus.

absence of adhesions and of metastases (as far as could be determined) made the case a favourable one for pneumonectomy.

(Case under the care of Mr. Tudor Edwards.)

Case 2.—**Carcinoma of Lung masked by Empyema.**

C. S., male, aged 64, a school caretaker. Four and a half months before admission he had ad "influenza" followed by cough and sputum which was occasionally bloodstained. For 1 month he had complained of left pleural pain, and 5 days before admission he became ill with pain in the left chest and fever. Examination: An ill, somewhat wasted man with clinical signs of a left basal pleural effusion (*Fig.* 196). Aspiration yielded slightly turbid seropus which gave a growth of pneumococcus on culture. After affording temporary relief by aspiration, intercostal drainage was provided, and later when the pus was thicker, rib resection was performed and closed drainage instituted. The general condition improved after drainage, but the lung failed to re-expand and sputum persisted. Sputum examination revealed the presence of carcinoma cells (probably columnar). A bronchogram confirmed this diagnosis by demonstrating a typical 'rat-tail' obstruction of the left lower lobe bronchus (*Fig.* 197). Bronchoscopy later showed growth in this bronchus and a biopsy was

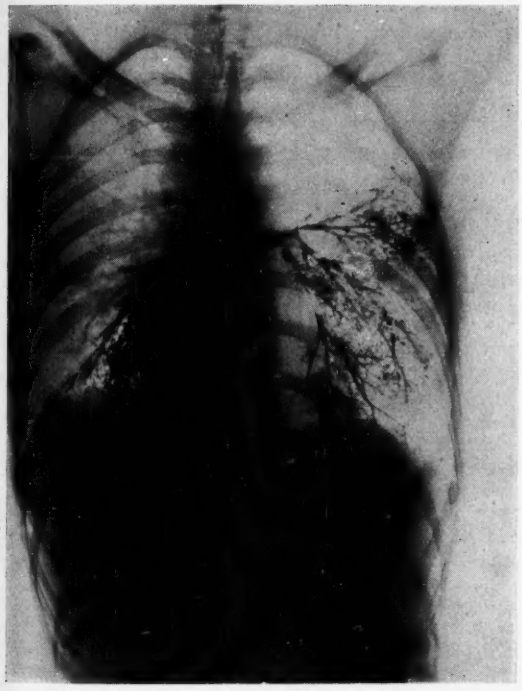

FIG 198.—*Case* 3. Bronchogram showing widening and deformity of tracheal bifurcation, and narrowing of right main bronchus. None of the opaque oil has entered the right upper lobe, but neither lung is filled adequately. The mediastinum is displaced to the right, and there is patchy collapse in the right upper zone.

reported polygonal-cell carcinoma. The stricture was dilated and radon inserted by means of a Tudor Edwards container. Some improvement followed, the empyema cavity became smaller, and the patient was discharged with a tube in situ. Three months later his doctor reported that he was becoming worse, was bedridden, and there was a profuse bloodstained discharge from the tube.

Here the initial symptoms of the disease were caused by a complication which at first masked the primary condition. Sputum examination provided the first evidence of the presence of a growth.

(Case under the care of Dr. R. A. Rowlands and Mr. Tudor Edwards.)

Case 3.—**Carcinoma of Lung diagnosed by Sputum Examination.**

H. K., female, aged 41. For 6 months there had been persistent cough and sputum, loss of weight, night sweats, and loss of appetite. During the 3 months before admission dark congealed blood had been coughed up on three occasions. Examination: A thin woman; slight pallor. Chest: Heart apex beat displaced slightly to the right; diminished movement, impaired percussion note, and bronchial breath-sounds over right lung. The sputum contained fragments of undifferentiated (probably columnar-cell) carcinoma (*see Fig.* 193). X-ray examination of chest and bronchography: Mediastinum displaced to right; patchy collapse in right upper zone. Bifurcation of trachea broad and irregular; narrowing of right main bro chus. None of the opaque oil entered the right upper lobe, but neither lung was adequately filled (*Fig.* 198). Bronchoscopy showed broadening of the carina and an ulcerated growth involving it and both main bronchi. Biopsy was

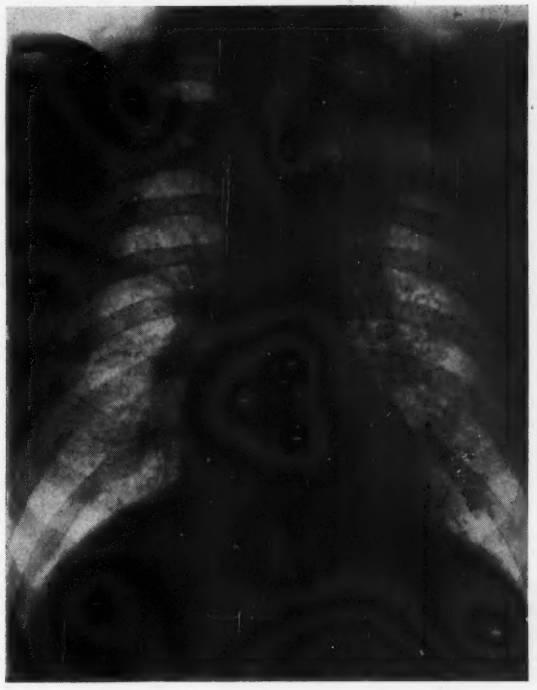

FIG. 199.—*Case* 4. Radiograph of chest showing opacity at the left apex. Residual neohydriol in lungs.

negative. The patient was considered unsuitable for any form of treatment and was discharged.

In this case no tumour was visible in the plain radiograph of the chest owing to the position of the growth, and there was no lobar collapse. The diagnosis was made in the first place by sputum examination and confirmed later by bronchoscopy.

(Case under the care of Dr. C. Wall and Dr. Russell Brain.)

Case 4.—**Carcinoma of Lung diagnosed by Sputum Smear: Bronchoscopy negative.**

G. D. T., male, aged 57, a lighter-fireman, complaining of pain in the lower left chest, made worse by deep breathing; some loss of weight. Examination: Nothing abnormal except scattered rhonchi in both lungs. X-ray examination: Shadow at left apex (*Fig.* 199). A bronchogram showed no filling in the area of opacity, but no obstructed bronchus was demonstrated. Bronchoscopy: Carina normal; no evidence of growth. Sputum examination showed fragments of squamous-cell carcinoma. Exploration of the chest was carried out and left pneumonectomy was performed with some difficulty owing to adhesions at the apex. The patient died 2 days later. The lung showed a carcinoma of the left upper lobe, and histological examination confirmed that it was a squamous-cell growth.

In this case the chief interest lies in the fact that sputum examination provided the only conclusive evidence of growth before operation.

(Case under the care of Mr. Tudor Edwards.)

ASPIRATION-BIOPSY

Another method of investigation which has been used to confirm the diagnosis of malignancy is aspiration-biopsy or lung-puncture. This method was first employed in a case of carcinoma by Ménétrier in 1886, and it has become popular in some American clinics within recent years. Craver and Binkley (1939) reported a series of 92 aspiration-biopsies in which positive results were obtained in 56 out of 62 (90·3 per cent) cases of proved or probable carcinoma. In 32 of these cases bronchoscopy was negative. It is claimed that the method causes little disturbance to the patient, that if it is done carefully with radiographic control it is practically devoid of risk, and that it is particularly valuable in peripherally-placed tumours. There is, however, a potential risk of disseminating carcinoma cells in the pleural cavity and the thoracic wall, and the method should be condemned if there is any hope of operating with a view to the removal of the growth. In view of the ease with which sputum examinations can be performed without any risk whatever to the patient, it would seem reasonable in any case to examine the sputum before submitting the patient to aspiration-biopsy. If used at all, this latter test should be reserved for establishing the diagnosis in cases giving repeatedly negative sputum tests, and in which operative removal of the growth is impracticable.

SUMMARY

1. The sputum from 93 cases of suspected neoplasm of the lung has been examined by Dudgeon's wet-smear method.

2. In a large proportion of cases (64·3 per cent of proved or probable carcinomata) it has been possible to demonstrate malignant cells in the sputum. It was not only in the advanced cases that such cells were found; in a significant proportion of the cases the growth was operable.

3. The test is of value in establishing the diagnosis of malignancy in cases where other methods of investigation, including bronchoscopy, have failed, and in which it is desired to explore the chest; in patients too ill to be submitted to other methods of investigation; and in cases where the growth is masked by secondary inflammatory changes in the lung.

4. It is urged that the test be employed more widely; this may lead to an earlier diagnosis being made in some cases.

5. It is further suggested that aspiration-biopsy as a method of diagnosis should be reserved for cases giving repeatedly negative sputum tests, and should not be employed when surgical removal of the growth is envisaged.

I am deeply grateful to Mr. Tudor Edwards, at whose suggestion this work was undertaken, for his constant encouragement and advice, and for his kindness and trouble in providing me with material from his hospital and private cases. I am indebted to Dr. P. N. Panton for providing laboratory facilities in the Pathological Department of the London Hospital; to Mr. N. R. Barrett for demonstrating the method to me personally, and for his helpful advice during the early stages of the investigation; and to Mr. John King of the London Hospital for the beautiful Dufay photomicrographs. The work was carried out while I was in receipt of a part-time grant from the Medical Research Council.

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TRAUMATIC CEREBRAL FUNGUS

By JOHN E. A. O'CONNELL

CHIEF ASSISTANT, SURGICAL PROFESSORIAL UNIT, ST. BARTHOLOMEW'S HOSPITAL; NEUROSURGEON, E.M.S.

THE formation of a cerebral fungus at the site of a penetrating wound of the brain is not infrequent. Of 38 patients with such wounds who were consecutively admitted to the neurosurgical unit of an E.M.S. hospital 11 (28 per cent) developed this complication either before or after their admission. A clear understanding of the condition is, therefore, essential in the treatment of the head injuries of warfare, since these are so frequently associated with cerebral penetration. The present study is based on the observation of the 11 cases mentioned.

The clinical features of brain fungus, the process of its healing, and the treatment of the condition will be described. The factors which predispose to the occurrence of a fungus will be considered, and a suggestion will be made as to the actual mechanism responsible for its development. The sequelæ of cerebral fungus will be noted, and brief résumés of important features of the cases in the series will be set out in a table.

It must be pointed out that by cerebral fungus is meant a protrusion of brain tissue through a defect in its coverings—i.e., scalp, skull, and meninges. This at once distinguishes the condition from a cerebral hernia in which the protrusion of the brain tissue through a skull defect remains covered by scalp and perhaps certain of the meninges. The conditions are to be differentiated since in brain hernia no infection is present in the herniated tissue and indeed no structural alteration of any kind necessarily occurs in it, while in brain fungus infection of, and structural alteration in, the exposed tissue are inevitable.

CLINICAL FEATURES

Time of Development.—Penetrating wounds of the brain differ not only in the depth of the penetration, but also in the superficial extent of the injury. Small metallic fragments of high velocity may pass deeply into the brain through a wound of very small size. Such a wound will often heal spontaneously and rapidly, and at no time will cerebral tissue be exposed on the surface—that is, no cerebral fungus will form. On the other hand, when the trauma affects a wider area of the coverings of the brain, it will expose a greater or smaller area of nervous tissue in the base of the wound, and a brain fungus is thus constituted. When the superficial extent of the destruction of cerebral coverings is small, the pulsating cerebrum exposed will show little tendency to protrude into and through the defect in its coverings, and there will certainly be no tendency to progressive protrusion even in the absence of treatment. When the superficial extent of the wound is greater, however, protrusion of cerebral tissue into the wound occurs and will be progressive if not checked by suitable treatment. Six of the patients in the series had wounds of small extent, and in these the fungus showed no tendency to enlarge, but the remaining five, with more extensive wounds, developed progressive fungation. It is of interest, however, that the tendency to such progressive enlargement of the fungus is not present from the first. Magnant (1927) states that it generally becomes obvious between eight and ten days after the injury. Occasionally, he says its onset may be delayed for more than two weeks, while in the presence of a very severe spreading infection of the brain it may arise as early as the third day after the injury. In the present series the fungus was frequently established at the time of the patient's admission, so that it is difficult to form an accurate estimate of the average interval between the receipt of the injury and the onset of progressive fungation; on the whole, it would seem that it occurs usually after an interval of from seven to fourteen days.

From what has been said it will be seen that other factors than the adequacy of treatment influence the occurrence of fungation; the more important of these are the type of wound and the interval between its production and the institution of treatment. It is believed that without full information on such points little benefit can follow comparison of the frequency of incidence of the complication in different groups of cases. In the present series the frequency of fungus formation is high for various reasons, of which the most important has been unavoidable delay in instituting treatment of the wound.

Clinical Signs.—In its early stages the protrusion will consist of pale soft pulsating cerebral tissue. Flakes of lymph and superficial sloughs adhere to it, and at this stage its appearance is in marked contrast with that of the healthy granulation tissue in the margins of the scalp wound. This surrounds the fungus and at a later stage becomes adherent to it. With the passage of time the appearance of the fungus changes. Its surface becomes clear of sloughs and takes on a deep red colour similar to that of the granulation tissue around it. At the same time the tissue becomes more rigid and resists deformation. As this change progresses the tendency for protrusion of the fungus to occur diminishes. Epithelialization slowly occurs from the margins of the wound, so that eventually a dense scar

covered by epithelium alone remains to indicate the previous site of the fungus.

The presence of a small cerebral fungus has been unassociated with any signs of systemic disturbance during the period of observation. It should be mentioned, however, that in two of the six cases of this type attempts had been made to suture the scalp over infected brain tissue; in both of these there was evidence of the spread of the infection in the cerebrum. This took the form of a raised temperature and pulse-rate and neck rigidity, together with an increase in the focal signs resulting from the wound; one of the patients had a series of generalized convulsions. With the separation of the margins of the scalp wound and the establishment of free drainage from the surface of the fungus, these signs subsided. It is believed that in each of the patients there must have occurred an infective encephalitis, infection spreading from the fungus into adjacent cerebral tissue. In both of them it appears likely that the encephalitis was of non-suppurative type.

Of the five patients with large cerebral fungi, three showed evidence of a systemic reaction. This again took the form of an increase in temperature and pulse-rate and signs of meningeal irritation, including an inflammatory reaction in the cerebrospinal fluid. The causation of these attacks is probably similar to that suggested to explain similar symptoms arising when the scalp is sutured over infected cerebral tissue. Although it has not occurred in any of the cases in the series, it is likely that sometimes the spread of infection from the fungus to neighbouring tissue is a more serious occurrence, so that either localization does not occur at all or it may occur with the formation of a cerebral abscess. It should further be pointed out that since all the patients received sulphanilamide or sulphapyridine by mouth, and local sulphanilamide was used in addition in some, it is likely that the incidence of these infective symptoms has been reduced and their danger diminished.

The occurrence of signs of focal nervous disturbance in association with cerebral fungation will depend, of course, upon the relationship of the wound to an area of the cerebrum of such functional significance that the resulting physiological defect is clearly demonstrable by clinical tests. The depth of the wound will also be important in determining the occurrence of focal signs, and, of course, at times these may be due to an associated injury and unconnected with the cerebral fungus itself. In 8 of the 11 patients focal signs were present, and in 7 these signs were of considerable severity.

Cerebrospinal Fluid.—The composition and pressure of the cerebrospinal fluid is of interest in cases of cerebral fungus. As regards pressure, in the only patient admitted within forty-eight hours of being injured the intracranial pressure was 200 mm. of cerebrospinal fluid in the early stages, and in subsequent weeks varied between 110 mm. and 210 mm. cerebrospinal fluid. In the other patients the pressure also varied from day to day, being at times between 100 mm. and 180 mm. cerebrospinal fluid, i.e., within the limits of normal, but at times above 200 mm. In no case was a severe or persistent intracranial hypertension revealed. Perhaps of greater interest than the actual cerebrospinal fluid pressure are some observations on the rachidian quotient—that is, the relationship between the volume of fluid withdrawn at the time of lumbar puncture and the resulting fall in pressure. In the early post-traumatic period the removal of a small quantity of cerebrospinal fluid (e.g., 20 c.c.) results in a marked fall in intracranial pressure. Later, as the protrusion develops, a considerably larger volume of cerebrospinal fluid (e.g., 100 c.c. or more) must be withdrawn to produce a similar fall in pressure. Later still, as the size of the fungus diminishes with healing, the volume of fluid which must be withdrawn to produce a given fall of pressure again becomes reduced. These observations suggest that when the fungus shows its maximum development there is an increase in the amount of cerebrospinal fluid in the cerebrospinal spaces. As will be indicated later, it is believed that this increased volume of cerebrospinal fluid is accommodated largely in the lateral ventricle of the injured cerebral hemisphere.

The cerebrospinal fluid is free from abnormality in some cases of fungus, but often there is evidence of a definite inflammatory reaction—a pleocytosis and a raised protein content. The pleocytosis has been largely lymphocytic in some instances, and it is of particular interest that at times the protein content of a specimen of cerebrospinal fluid has been greatly increased (300 mg. per cent) without there being any degree of plecytosis present in the fluid. It would seem that the free drainage of exudate to the exterior from the surface of a cerebral fungus may render a spreading encephalitis less common than is the case in the presence of a closed cerebral infection—and in this way the occurrence and nature of the meningeal reaction in fungus and abscess differ. The high protein content of the cerebrospinal fluid may perhaps be due to the removal from the fungus through the ventricular wall of fluid rich in protein. It might be expected that such an exudate would be present in the fungus as the result of leakage from its blood-vessels and the degeneration of nervous tissue.

Pathological Anatomy.—It is surprising to find considerable discussion in the literature as to the exact constitution of a cerebral fungus. The earlier authors suggest that the whole of the fungus is composed of cerebral tissue. McEwen and others, quoted by Magnant, believed that a fungus is but in part composed of cerebral tissue, and suggested for it the designation 'pseudo-hernia of the cerebrum'. Leriche described two essential parts of the fungus—a superficial shell

of organizing clot and granulation tissue and a deeper portion composed of partially disorganized nervous tissue. Magnant mentions the adhesions which bind together the dura, arachnoid, and pia mater around the margin of the fungus, and states that, while they, of course, form a defence against infection of the meninges, they act as a collar around the neck of the protruding cerebral tissue, which favours obstruction of its venous return and the growth of the fungus. He further suggests that in the stage of healing the subarachnoid space is reconstituted between the fungus and the overlying scar—in other words, that the brain becomes covered by reconstituted meninges in the traumatized area.

The following description is based on clinical observation as no post-mortem material has been available for accurate anatomical study. A penetrating wound of the type which produces brain fungus results in destruction of an area of scalp, skull, meninges, and brain. Following such a wound, adhesions between pia and dura mater around the margins of the injured area will usually soon seal off the subarachnoid space, thus preventing a suppurative meningitis. The exposed cerebrum will be pale, soft, and œdematous, with adherent blood-clot and sloughs.

At this early stage there will be little tendency to protrusion. Later, for reasons which will be explained, protrusion of the exposed cerebral tissue occurs. Gradually it will be noticed that this tissue changes its macroscopic characteristics, becoming red in colour and firm in texture. It is believed that these alterations are due to the development of granulation tissue. This is probably brought about by the development of new blood-vessels and hyperplasia of the perivascular connective tissue. Perhaps there is also an ingrowth of vessels from the scalp margin which tends to adhere to the fungus. Epithelialization of the fungus can only occur when this bed of healthy granulation tissue has formed.

It appears, therefore, that healing commences with the formation of granulation tissue, but that this process takes longer than it does in other parts. This may be due to the peculiar structure of nervous tissue, but whatever its cause this delay in the healing process is believed to be an important factor in permitting the development of a cerebral fungus. When epithelialization has occurred it is believed that the scar beneath the epithelium, adherent to the edges of both bone and meningeal defects and extending deeply into the cerebrum, is permanent. In other words, there is no attempt at reconstitution of the meninges such as has been suggested by some authors. These stages in the development and healing of a cerebral fungus are represented in the diagramatic drawings, *Figs.* 200–202.

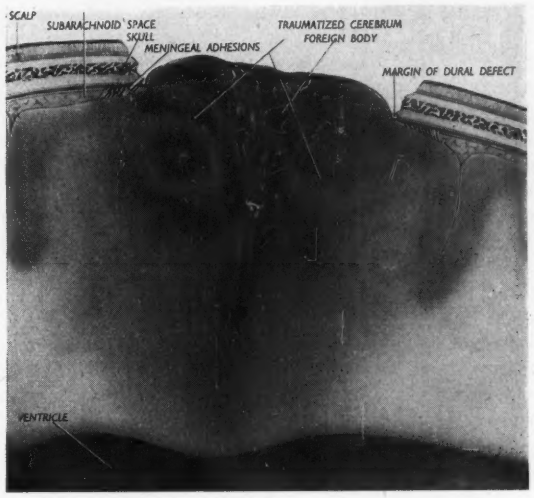

FIG. 200.—Drawing representing the anatomy of a penetrating wound with considerable destruction of the scalp and skull. The stage represented is the early post-traumatic period.

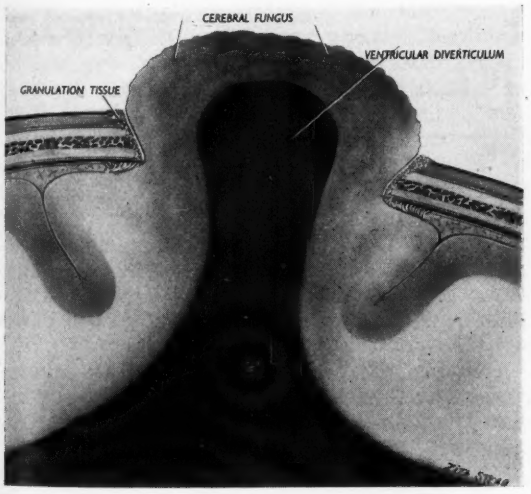

FIG. 201.—Drawing representing the anatomy of a fully developed cerebral fungus.

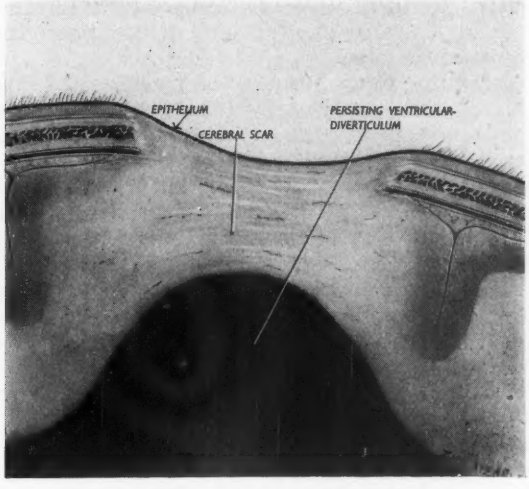

FIG. 202.—Drawing representing the injured area after healing of a cerebral fungus.

AETIOLOGY

The previously Accepted Theories.—There has been much difference of opinion as to the mechanism which is responsible for the production of a cerebral fungus. Magnant has classified the aetiological factors which have been suggested into two groups—infective and mechanical. The infective hypotheses include localized encephalitis, brain abscess, and retained foreign bodies, the suggestion being that in the presence of one or more of these the intracranial pressure is raised and that as a result protrusion of the brain occurs through the superficial defect. It has already been pointed out that in many instances intracranial pressure is not raised in the presence of a large fungus; further, since an abscess or retained foreign body will often be absent, it must be admitted that the infective hypothesis does not provide an adequate explanation for the development of the majority of cerebral fungi. It might be thought that inflammatory swelling of the injured and infected cerebral tissue in the absence of abscess or a foreign body might be responsible for the development of the fungus. It is difficult to see why the protrusion should not begin to appear for one or two weeks after the injury if this were so. It would be expected that inflammatory swelling would be maximum in the early post-traumatic period and would subsequently diminish. The fungus thus makes its appearance and enlarges at a time when œdema resulting from trauma or a freely draining infection should have largely disappeared. Stanley (1820) stresses this point in a paper written over one hundred and twenty years ago and makes other pertinent observations. Thus infection may be a contributory factor; it is certainly not the essential one.

Turning to the mechanical hypothesis, Lamey and Flourens, quoted by Magnant, suggest that the condition is due to the expansion of the cerebrum produced by the pulsation in its arteries. It is difficult to see why the pulsation in the cerebral vessels should be greater than normal, and if this be not so it cannot be sufficient to carry the cerebrum through the dural opening for many centimetres. Nélaton, quoted by Magnant, has suggested that the loss of cerebral coverings is the cause, but this explanation is not adequate when it is recalled that intracranial pressure is, in the horizontal position, but 10 cm. of water above atmospheric pressure and with the head of the bed raised may be subatmospheric. It may be pointed out that such postural treatment will not control the development of a fungus. Again, it has been suggested that obstruction to the cerebrospinal fluid circulation may be the causative factor; adhesions in the neighbourhood of the roof of the fourth ventricle (due to inflammation) could produce such obstruction. The arguments which make such a hypothesis untenable will be given later. Finally, the hypothesis of Leriche (1916), which has been widely accepted, must be considered. Leriche suggests that as a result of œdema developing in the contused brain it exteriorizes itself through the breach in its coverings, and that the protruded tissue is then strangulated by the margins of the bony orifice which prevent the re-establishment of venous circulation; hæmorrhage then occurs into the strangulated brain and the fungus is constituted. Based on this hypothesis is its author's operation of enlarging trepanation for the cure of cerebral fungus. This consists in the removal of bone so as to enlarge the skull defect, the small size of which is regarded as the cause of the strangulation. In this way venous congestion is said to be relieved and regression of the fungus is permitted. There are several reasons why this hypothesis does not appear to be acceptable. In the first place, where the defect in the cerebral coverings is small, progressive fungation of the brain does not occur, although it might be expected that such small skull defects would more readily produce strangulation of the protruded brain than would larger ones. It is, of course, the latter which are associated with progressive cerebral fungation. In the second place it is believed that the superficial extent of the fungus is determined by the size of the dural and not that of the skull defect. Not infrequently it will be found that the dural breach is considerably smaller than that in the skull, and in such cases any strangulation of protruded brain which occurs must be produced by the dural edge. It is therefore felt that any benefit following the operation of enlarging trepanation is not due to the relief of strangulation produced by the bone edge. Another possible explanation of the benefits of this operation will be suggested later.

An Alternative Hypothesis.—It will be seen from the above discussion that there are strong arguments against the acceptance of current explanations of the mechanism productive of traumatic cerebral fungus. An alternative hypothesis will now be put forward, and it is believed that it explains the known facts in connexion with the condition. In the first place, there is always a localized dilatation of the lateral ventricle in relationship to a cerebral fungus. The evidence for this statement is of several kinds.

Roques and Huard (1937) illustrate and describe serial sections through the brain in a fatal case of the condition. These show clearly what must be a simple prolongation of the ventricle into the fungus, though the authors took it to be a cavity due to destructive encephalitis which formed a secondary communication with the ventricle. They seem to have assumed, like many others, that the ventricle was drawn into the fungus and therefore came to communicate with this cavity. In discussions on the treatment of the condition, the danger of attempted excision of the protruded brain has frequently been stressed, and it is stated that the risk of such a

procedure lies in the likelihood of opening a prolongation of the lateral ventricle into the fungus.

Had it been considered justifiable to carry out an air injection (ventriculography or encephalography) in all the cases of fungus observed, the truth of the statement that the ventricle itself protrudes into the fungus would have been clearly demonstrated. Such an investigation was not done, since it did not appear to be in the interests of the patient during the stage of the developed fungus. One of the patients had an epileptic attack six months after his injury, and was, therefore re-admitted and encephalography carried out. The films (*Figs.* 203, 204) clearly show dilatation of the frontal horn of the left lateral ventricle towards the bone defect in the left frontal region. Although when these films were made the fungus had disappeared, it is considered that the dilatation was present from an early stage, as in the case of Roques and Huard referred to above. In other words, it is believed that the dilatation is not a late effect due to traction of scar tissue on the ventricular wall.

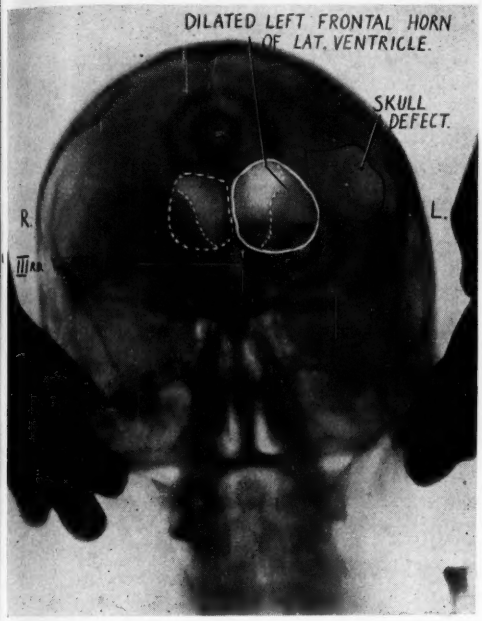

FIG. 203.—W. J., gunshot wound of left frontal region. Encephalogram—antero-posterior.

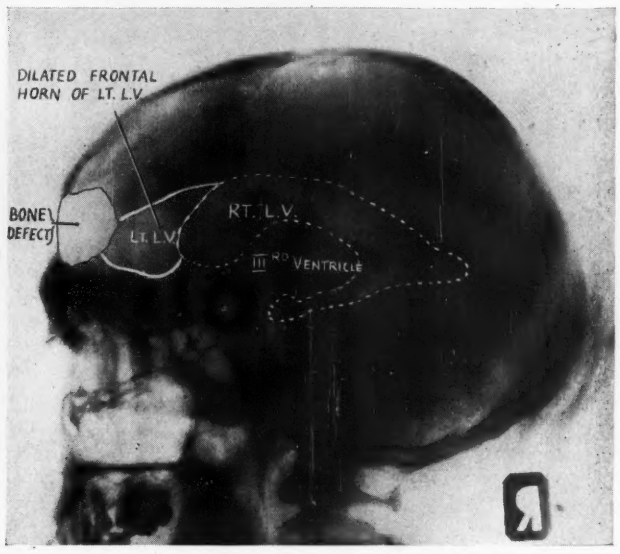

FIG. 204.—Same case. Encephalogram—lateral, left side uppermost.

It has already been mentioned that on lumbar puncture the volume of fluid which must be removed to reduce the fungus increases as the fungus enlarges, and diminishes as it subsides. It is believed that the additional volume of fluid is contained in the dilated portion of the ventricle. The striking effect of lumbar puncture in reducing a fungus of considerable extracranial extent to a concavity, immediately suggests that a strangulation of the extruded brain is not the causative factor, but that fungus formation is dependent on the amount of cerebrospinal fluid in the ventricles.

The ventricular dilatation which occurs in relationship to a cerebral fungus is not unexpected when it is borne in mind that such dilatation frequently occurs when cerebral tissue is injured. It is common knowledge that when a cerebral abscess is tapped, the neighbouring portion of the ventricle undergoes rapid dilatation. If the abscess is deeply placed, subsequent attempts to tap it along the same track may open the ventricle. The encephalogram (*Figs.* 205, 206) of a patient with a cerebral abscess illustrates this point. A large recurrent temporosphenoidal abscess had been drained and the tube had been in place for several weeks. As the abscess cavity diminished in size the lateral ventricle enlarged. Another demonstration of the occurrence of this localized internal hydrocephalus can be seen in the films (*Figs.* 207, 208) of a soldier who sustained a severe closed head injury with a fissured fracture in the left occipital region. After a long period of unconsciousness this patient slowly recovered from a severe left hemiparesis which was doubtless the result of a *contre-coup* lesion in the right temporofrontal region. Encephalography carried out some months after the injury shows marked dilatation of the frontal and temporal horns of the lateral ventricle in relationship with the injured area of cerebrum.

From the evidence put forward above, it is believed that localized ventricular dilatation frequently occurs in relationship with an area of injured cerebrum. Further, it is believed that before progressive cerebral fungation can occur such ventricular dilatation must take place, and, indeed, it is the progressive ventricular dilatation which leads to the progressive cerebral fungation.

The cause of the localized ventricular enlargement must now be sought. In the first place it is frequently suggested that the dilatation of the ventricle occurring in relationship with injured by a cerebral cicatrix does contribute to the ventricular dilatation in its later stages, it is considered that it plays no part in the dilatation which occurs at an early stage, and which is seen

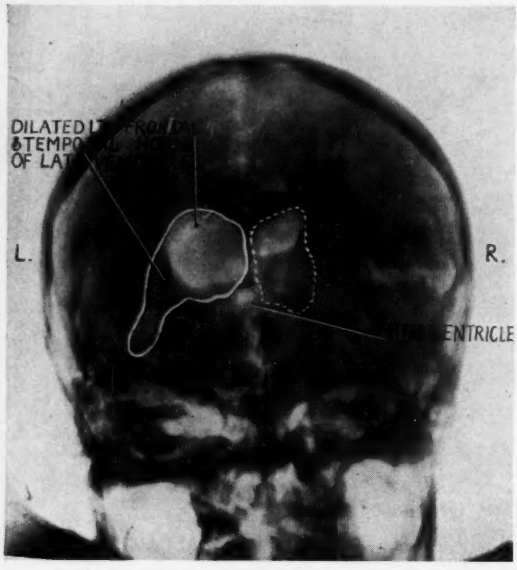

FIG. 205.—J. N. Left temporosphenoidal lobe abscess. Encephalogram—antero-posterior (film reversed in course of reproduction).

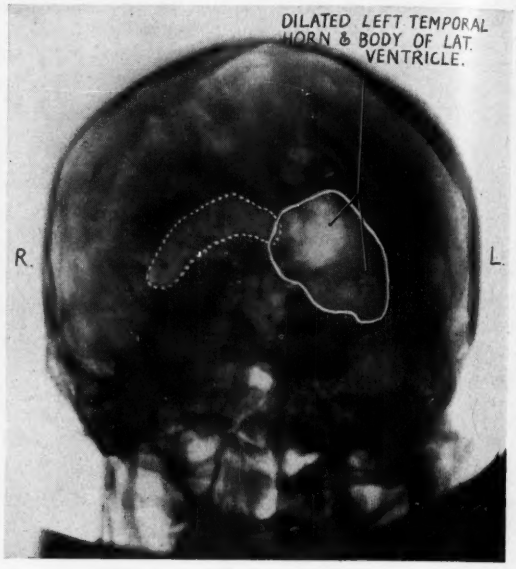

FIG. 206.—Same case. Encephalogram—postero-anterior (film reversed in course of reproduction).

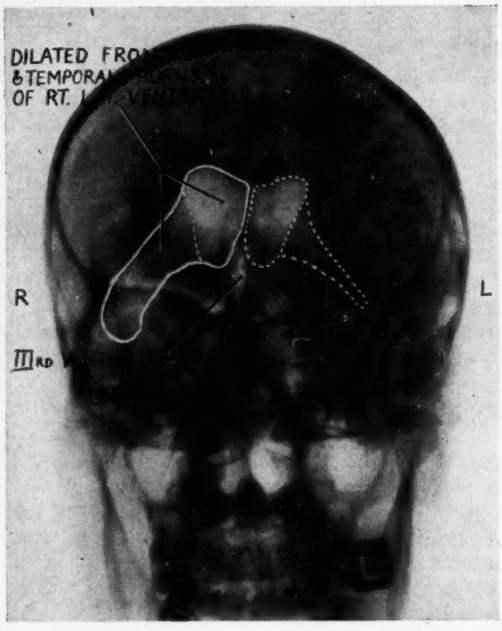

FIG. 207.—G. B. Fissured fracture left occipital region. Encephalogram—antero-posterior.

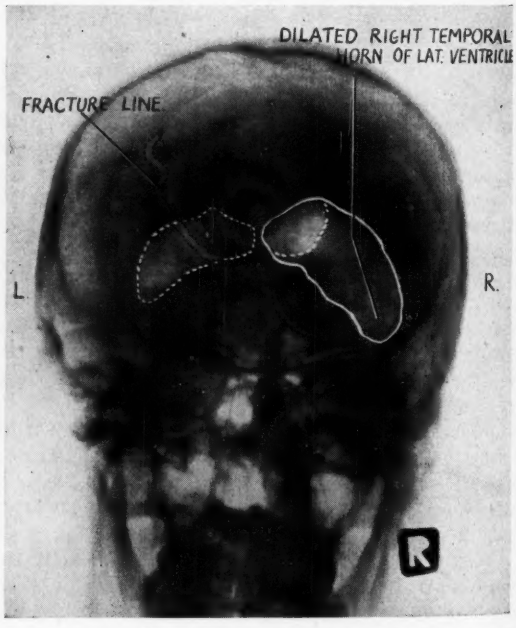

FIG. 208.—Same case. Encephalogram—postero-anterior.

cerebrum is due to traction of an overlying scar—the appearance in an encephalogram being referred to as 'tenting'. Penfield and Foerster (1930, 1934, etc.) frequently refer to the appearance. While it may be that such traction produced in the examples quoted above. Indeed, it can have little bearing at any time on the dilatation of the ventricle which occurs in relationship with a drained cerebral abscess or an area of cerebrum involved in a closed head injury. In the second

place, it might be suggested that the localized ventricular enlargement results from an obstruction to the cerebrospinal fluid circulation produced by an inflammation of the meninges. Several arguments can be put forward against such a view. The ventricular dilatation is a localized one and not a diffuse internal hydrocephalus as would be expected if such obstruction to the circulation existed. The ease with which a large volume of cerebrospinal fluid can be drained from the ventricles suggests that no obstruction to the escape of fluid from the ventricles exists. The immediate rise in lumbar pressure which follows gentle pressure on the fungus has a similar implication. The cerebrospinal fluid frequently shows no evidence of an inflammatory reaction even in the presence of a large fungus. For these reasons it is considered that obstruction to the cerebrospinal fluid circulation is not the cause of the localized ventricular dilatation occurring in association with a cerebral fungus or the other pathological processes mentioned above. It is believed that intraventricular pressure acting on an area of ventricular wall which has been damaged by direct injury, pressure, or vascular lesions, is the responsible factor in the conditions under consideration, and that it is thus the most important factor in the production of progressive cerebral fungation.

It is now generally agreed that the pressure in the capillaries of the choroid plexus, and perhaps the vital activity of its epithelium, are responsible for the production of cerebrospinal fluid, and further, that the absorption of the fluid into the venous system is largely due to the osmotic attraction of the blood proteins, and, to a lesser extent, to the difference between the higher cerebrospinal fluid and lower venous pressure on the two sides of the arachnoid villous endothelium. Were these the only factors to be considered, the circulation of fluid would amount to the addition of fluid to the total bulk of cerebrospinal fluid at one site and its subtraction at another situation, the actual circulation between the points of production and absorption being purely passive. From certain clinical observations it is believed that the circulation is a much more active one than this, and is comparable with, and indeed dependent upon, the circulation of the blood.

With every cardiac systole there is a definite increase in the amount of intracranial blood proportional to the expansion of the cerebral arteries. It is obvious in the pulsations (or increases in volume) of the brain which are visible when it is exposed. It is seen, too, in the considerable variations in intraventricular pressure which can be observed to occur with each heart-beat. It is seen again in the intermittent escape of cerebrospinal fluid which occurs when a large cerebral subarachnoid channel is punctured. This intermittent flow corresponds in its rhythm with the pulse rhythm. In normal circumstances it is believed that these phenomena point to rapid circulation of at least the cranial fraction of the subarachnoid fluid, which is therefore regarded as being an active process only indirectly affected by the rates of production and absorption of the fluid. The latter factors largely control what might be called the cerebrospinal fluid volume. Like the blood-volume, it only indirectly affects the rate of circulation of the fluid. The circulation of the blood is dependent on cardiac contractions for its occurrence, and it may well be that the circulation of the cerebrospinal fluid is similarly dependent.* What will be the effect of the rise in intraventricular pressure which occurs during cardiac systole upon a damaged area of cerebrum?

In ordinary circumstances when ventricular volume diminishes and intraventricular pressure rises during this stage of cardiac activity, it is believed that the increased pressure expels fluid from the lateral and third ventricles through the aqueduct into the fourth ventricle and through the foramina in the roof of this ventricle into the subarachnoid space. In the presence of diminished resistance of part of the ventricular wall, this increase in pressure during systole will be expended partly in its normal expulsion of fluid from the ventricle and partly in the production of dilatation of the lateral ventricle into the damaged area. The latter will continue until the ventricular dilatation has made good the loss of cerebral tissue and all parts of the ventricular wall again present an equal resistance to the systolic increment of pressure. In cases in which the cerebral injury is associated with the loss of a considerable area of scalp, skull, and dura, the systolic rise in intraventricular pressure will not meet with normal resistance when the cerebrum has gained the level of the dural defect. Further enlargement of the ventricle will therefore occur, and the cerebrum will be carried through the defect in its coverings to form a protrusion. If untreated this protrusion will tend to be progressive, since as the ventricular wall thins, its resistance to the intraventricular thrust will diminish. The intraventricular pressure will, of course, remain undiminished, since, as ventricular volume increases, fluid is retained in the ventricle, and therefore no fall in pressure occurs. In considering the force responsible for ventricular dilatation, the importance of the systolic rise in intraventricular pressure has been emphasized. It should be further added that there is a slower but greater excursion of intraventricular pressure with the phases of respiration and dependent on the fluctuating volume of the intracranial venous blood. This is a factor of probably equal importance in the circulation of the cerebrospinal fluid and the development of localized ventricular dilatations.

It has already been pointed out that there are two types of cerebral fungus. When the defect in cerebral coverings is of small extent protrusion of brain through the dural defect will be slight or

* It is hoped to discuss fully the evidence for this statement in a subsequent communication.

absent. Such protrusion as occurs is regarded as being due to œdema in the injured brain. It will show no tendency to progression because of the resistance offered by the small dural opening to the passage through it of further cerebral tissue containing a ventricular prolongation. On the other hand, when there is a more extensive loss of cerebral coverings, progressive fungation will occur. It has already been stated that in many cases this does not take place for a week or more after the receipt of the injury. A possible explanation of this fact lies in the occurrence of œdema in the injured brain. In the early post-traumatic period this will give rise to an increase in intracranial pressure. The ventricle in the neighbourhood may be somewhat narrowed and the dural defect effectually plugged by the swollen brain tissue. As this œdema subsides the mechanism above described comes into action. The state of affairs at the early stage is represented in *Fig.* 200. The developed fungus is represented in *Fig.* 201.

Two important factors in the development of a cerebral fungus have been discussed—the defect, and the force which causes the cerebrum to protrude through it. A third factor must also be mentioned—the resistance offered to the occurrence of protrusion. The essential nature of nervous tissue with a low content of adult fibrous tissue endows it with relatively little resistance to deformation. A wide area of uninfected brain can be covered by a scalp flap, and if this is adequately sutured in position, the tendency for the brain to protrude is readily controlled by it. With the developed fungus in its early stages it is surprising how quickly a large protrusion re-forms after reduction by lumbar puncture has been brought about. Later, as the brain tissue becomes converted into granulation tissue, such rapid fluctuations in size of the fungus cease, and eventually the tissue develops such rigidity that it is no longer affected by variations in intracranial pressure and protrusion ceases. With epithelialization of the granulation tissue the wound is healed as indicated in *Fig.* 202.

To summarize this hypothesis it may be stated that when a breach in the cerebral coverings is of sufficient extent, ventricular dilatation (which always occurs into injured brain because of the increments in intraventricular pressure during a cardiac systole and expiration) will be unresisted, and will carry the cerebrum through the dural defect to give rise to a cerebral fungus. If the fungus is controlled the injured cerebrum eventually becomes converted into granulation tissue of sufficient rigidity to resist variations in intracranial pressure, and protrusion no longer occurs. With epithelialization of the area healing is complete.

TREATMENT

Before considering the treatment of a fully developed cerebral fungus it should, perhaps, be emphasized that this complication of a head injury is, in most instances, avoidable. Early surgical treatment of a penetrating wound will prevent its development. Such treatment should consist in excision of the scalp edge, which need never be extensive, excision of the margins of the bone defect *en bloc* and the removal of devitalized brain, blood-clot, and foreign bodies from the cerebral wound according to the method described by Cushing. Accurate suture of the scalp wound in two layers should then be carried out whenever possible. If the destruction of scalp be too extensive to permit this, it may be possible to cover the exposed cerebrum with a scalp-flap, while a Thiersch graft is placed over the denuded area of cranium. Lumbar puncture is advisable at intervals after such operations in order to relieve the suture line from tension, and thus prevent the development of a fungus which would be inevitable if primary healing of the scalp wound did not occur.

When a cerebral fungus is established the treatment required will vary with the type of fungus. After a wound which has produced a small loss in cerebral coverings, cerebral tissue will constitute the base of the ulcer, but will show no tendency to protrude progressively. In such a case the state of affairs differs little from that of a traumatic ulcer elsewhere, except that the slow conversion of cerebral tissue into granulation tissue prolongs the process of healing. The treatment of a fungus of this type differs from that of other ulcers only in so far as there is a danger of infection of the meninges from the wound, and in that every precaution must be taken to avoid further injury to nervous tissue by mechanical or chemical trauma. To these ends a course of chemotherapy with one of the sulphonamide group of drugs has been given in each case. In some, local chemotherapy has also been employed. A non-irritating and non-adherent dressing has been applied to the wound —oiled silk, gauze soaked in flavine and paraffin, or gauze impregnated with vaseline—the last being, perhaps, the most satisfactory.

Cerebrospinal Fluid Drainage.—When the breach in the superficial tissues is more extensive the risks of meningeal infection and of increasing the extent of the cerebral injury are combated as described above in the case of smaller wounds. But in these larger wounds progressive fungation of the cerebral tissue occurs and it is of the greatest importance that it be restrained, since it increases the damage to the cerebrum and the risk of infection of the ventricular cavity. As has been explained above, it is believed that the progressive fungation is due to progressive dilatation of the underlying lateral ventricle, and that the latter is due to variations in intraventricular pressure. By reducing the quantity of cerebrospinal fluid in the ventricles and cranial subarachnoid space, this ventricular dilatation can be prevented or minimized. Such reduction can be brought about by posture, control of the fluid intake, and by the withdrawal of cerebrospinal fluid. When

the head of the patient's bed is raised on 12-in. blocks intracranial (and intraventricular) pressure is lowered and the force causing protrusion is thus lessened. By maintaining the fluid intake at 1500 c.c. per 24 hours, mild dehydration may be produced and a slight lowering of intracranial pressure results; dehydration can be further encouraged by saline purgatives and magnesium sulphate enemata.

Of much greater importance, and, indeed, the essential part of the treatment of progressive fungation, is cerebrospinal fluid drainage. This is usually carried out by lumbar puncture, but in one patient in the series ventricular drainage was considered necessary. When lumbar drainage is employed it is essential that the procedure be carried out at sufficiently frequent intervals, and that an adequate volume of fluid be removed on each occasion. In one patient control of the fungus necessitated lumbar puncture forty-five times; at first this procedure was carried out at daily intervals, but as the structure of the injured brain became more rigid the interval was increased to two days or more. In this patient as much as 150 c.c. of fluid was frequently withdrawn during the course of a lumbar drainage, and during the whole period of such treatment over four litres of fluid were removed from the subarachnoid space. The indication for lumbar drainage will be the presence of a protrusion of any extent, and drainage will be maintained on each occasion until the protrusion becomes converted into a deep concavity. This will usually entail lowering intracranial pressure to a low figure—often to zero in the horizontal position. If this lowering of intracranial pressure is produced rapidly it will often occasion very severe headache, profuse sweating, vomiting, and drowsiness. Instead of the intermittent rapid drainage it has been our practice to provide a more prolonged and slower flow by leaving the lumbar-puncture needle in situ for several hours; by this method these symptoms may be diminished or avoided altogether.

In one patient—an early case—in whom lumbar puncture did not adequately maintain reduction of the fungus, a catheter was inserted into the opposite lateral ventricle and continuous ventricular drainage maintained for six days. It was found that this did not prevent the protrusion of the cerebrum, although the fungus remained smaller than it had been prior to the institution of ventricular drainage. This is, no doubt, to be explained by the fine catheter offering a greater resistance to intraventricular pressure than did the weakened area of ventricular wall beneath the skull defect. It is now considered that this procedure is probably almost always unnecessary, and that lumbar drainage repeated as frequently as is indicated is the treatment of choice.

Magnant produces arguments against the use of lumbar puncture in cases of cerebral fungus. He states that it is dangerous in that it produces a rapid decompression, that it may lead to meningitis, and that it is anyway not proven that cerebral fungus is due to intracranial hypertension. None of these arguments is applicable for reasons that have been discussed already.

Special Methods of Local Treatment.—While it is believed that the measures described above—prevention of infection of meninges, protection of cerebrum from further injury, and the maintained reduction of the fungus by cerebrospinal fluid drainage—are the essential features of the treatment, it is considered that some other minor points deserve mention. Firstly, it will be noticed in the course of lumbar puncture that at times the central part of the fungus alone recedes; its margin has become adherent to the granulation tissue covering the scalp and skull margin (*see Fig.* 201). Careful separation of these soft adhesions by stroking with moist cotton-wool will allow of a more complete reduction of the fungus than would otherwise have been possible. The benefit of the lumbar drainage wlil be prolonged in this way. It is obvious that any such manipulation must be carried out with the greatest care, in order that the meningeal spaces be not laid open and the cerebrum be not further injured.

Secondly, since fungation occurs because of the lack of resistance of the injured ventricular wall to variations in intraventricular pressure, and ceases when this wall has developed sufficient rigidity by being converted into firm granulation tissue, the question arises whether the force producing protrusion could be successfully resisted at an earlier stage. It has been found that if the concavity (which replaces the protrusion after cerebrospinal fluid drainage) be lightly packed and covered by a firmly bandaged dressing recurrence of the protrusion is delayed. In some cases the packing was of gauze tape soaked in flavine and paraffin, and in others gauze tape separated from the cerebrum by oiled silk. In one patient a piece of stent was moulded to such a size and shape that it could be used to form a rigid covering over the skull defect. It was fixed to the head with adhesive tape after a dressing had been applied. This, too, restrained the growth of the fungus. When lumbar puncture is performed as often as it is indicated such a measure as the latter will rarely be necessary. Should the ventricular wall be greatly thinned it might even be dangerous, since pressure necrosis and a ventricular fistula might result, with all its attendant risks of infection of the cerebrospinal spaces.

Thirdly, there are reports of success with applying a skin-graft to a cerebral fungus. In one patient in the series a graft was applied in the early stages, but failed to take. In most cases the procedure has nothing to recommend it, since the granulation tissue and the surrounding meningeal adhesions provide a most satisfactory barrier to the spread of infection, and a skin-graft is not required from this point of view. If the graft is applied too early, that is, before a good base of granulation tissue is present, it is liable to fail—the more so because the pulsation

of the cerebrum makes it difficult or impossible to prevent movement of the epithelium on the underlying brain. At a later stage, when healthy granulation tissue covers the surface, epithelialization of the area from its margins will usually be rapid, and a graft is therefore unnecessary. In a very extensive loss of superficial tissue it might be that a Thiersch graft would be of value, in that epithelium over the centre of the area would form late and might be liable to ulceration, Sometimes as hair grows up around the epithelialized scar its ends may excoriate the epithelium, which may require protection by a small gauze pad for some weeks after epithelialization has been completed.

Other methods of treatment used in the past —the application of caustics to the surface of the fungus, ignipuncture and galvanopuncture, reduction by force, and ablation have no place in modern therapy. It is considered that Leriche's operation of enlarging trepanation is founded on an incorrect conception of the aetiology of a cerebral fungus. It is the margins of the dural defect (*see Fig.* 201) which form the orifice through which fungation occurs. The removal of skull from around its margins, leaving the dura intact, can therefore do nothing to relieve a hypothetical venous congestion in the fungus. Further, it has been suggested above that venous congestion due to strangulation is not a factor of importance in the aetiology of the condition. Thus, if the operation of enlarging trepanation does benefit a cerebral fungus, the benefit of the procedure must have some other explanation than that which has been put forward by Leriche. Possibly the systolic rise in intracranial pressure, instead of exerting its effect on the exposed cerebral tissue alone, will, after this operation, in addition produce a slight amount of protrusion of the area of dura around the fungus and over which bone has been removed. In this way the force tending to produce the fungus might be reduced to some extent. In any case it is considered that this operation is quite unnecessary in the modern treatment of cerebral fungus.

COMPLICATIONS AND SEQUELÆ

In the early stages of the development of a cerebral fungus there is a constant risk of infective complications. In this series there has not been a case of meningitis. One patient was found to have three cerebral abscesses in relationship

Table I.—CASES OF LARGE FUNGUS WITH PROGRESSIVE PROTRUSION

PATIENT AND AGE	TYPE OF INJURY	CONSCIOUSNESS	SIZE OF FUNGUS	FOCAL NEUROLOGICAL DISTURBANCE	AETIOLOGICAL FACTORS	INTERVAL BETWEEN INJURY AND PROTRUSION	DURATION OF PROTRUSION	CEREBROSPINAL FLUID ALTERATION	SEQUELÆ	TREATMENT
W. F. S., 20	G. S. W., right fronto-parietal	Not lost	9 × 2·5 cm.	Left hemiplegia	Extensive loss of scalp, etc. No attempt to cover denuded cerebrum	Admitted 10 days after injury with established fungus	5 weeks	Protein reaching 520 mg. per cent Lymphocytes reaching 127 per c.mm.	Left hemiplegia	Posture. Dehydration. Chemotherapy Lumbar drainage. Ventricular drainage Packing and stent mould
I. B., 11	G. S. W., left fronto-parietal	Lost	5 × 5 cm.	Right hemiplegia. Aphasia	Heavy infection of wound with hæmolytic streptococci Operation within 24 hr.	6 days	6 weeks	Protein reaching 120 mg. per cent Lymphocytes and polymorphonuclears each reaching 75 per c.mm.	Right hemiparesis	Posture. Dehydration. Chemotherapy Lumbar drainage. Packing
G. C., 25	G. S. W., right frontal	Not lost	7 × 4 cm.	Right hemiparesis	No treatment other than field dressing for 4 days	6 days	10 days	Protein rising to 200 mg. per c.mm. Cells rising to 250—35 per cent polymorphs; 65 per cent lymphocytes	Epilepsy	Posture. Dehydration. Chemotherapy Lumbar drainage
R. B., 25	Injured in collision at sea Compound depressed fracture right frontal	Lost	7 × 2·5 cm.	Nil	No attempt at treatment of wound for the first 10 days	Admitted 10 days after wound with established fungus	8 days after admission	Protein rising to 60 mg per cent No white blood-corpuscles	Epilepsy	Posture. Dehydration. Chemotherapy Lumbar drainage
D. J., 18	Buried in bombed building Compound depressed fracture left temporal	Lost	7·5 × 5 cm.	Right hemiparesis. Aphasia	Wounded eight days before débridement attempted	Admitted 10 days after wound with established fungus	4 weeks	Protein, 40 mgm. per cent No white blood-corpuscles	Slight residual expressive dysphasia	Posture. Dehydration. Drainage of cerebral abscesses. Chemotherapy Lumbar drainage

with a fungus. Following the drainage of these he was treated as were the other cases, and made a satisfactory recovery. In five patients one or more attacks occurred characterized by fever, a raised pulse-rate, drowsiness, and an increase in the focal signs due to the fungus. In addition there were signs of meningeal irritation, with an inflammatory reaction in the cerebrospinal fluid, although no organisms were found in it. These attacks passed and it is believed—though not proven—that they were the result of a non-suppurative focal encephalitis resulting from the spread of infection from the fungus into neighbouring nervous tissue. Possibly an abscess will be found in certain of these patients at some future date. This focal encephalitis develops early in the life history of the fungus; after a short period the granulation tissue barrier prevents its occurrence. It is probable that the incidence and severity of these infective complications have been considerably reduced in this series by the use of the sulphonamide group of drugs.

Two important and frequent sequelæ occur. Four of the eleven cases have developed *epilepsy*. Since none of the patients was injured more than two and a half years ago, it is probable that several others will in due course develop epilepsy. The dense nature of the cerebral scar (*see Fig.* 202) which replaces the fungus is doubtless related to the frequency of epilepsy, and it is possible that its site also influences the liability to this sequela, though the series is much too small to justify anything more than a tentative suggestion on this point.

The other important after-effect is, of course, *focal nervous disturbance*. Its presence or absence will depend on the site of the wound. Six of the eleven patients showed this sequela in varying degree. It is remarkable how signs of severe focal nervous disturbance in the early stages may disappear completely as the fungus heals. However, in so far as the focal signs are due to actual destruction of nervous tissue, recovery cannot be expected.

At one time cerebral fungus was associated with a very grave prognosis. In the literature the mortality in different series varies from nil to 70 per cent. It is difficult to evaluate the figures given. With non-progressive fungation recovery should usually occur without any special treatment. On the other hand, inadequate treatment of a large progressive fungus may lead to the death of the patient. In the present series there was no fatality.

Table II.—Cases of Small Fungus showing no Tendency to Progressive Protrusion*

Patient and Age	Type of Injury	Consciousness	Size of Fungus	Focal Neurological Disturbance	Aetiological Factors	Cerebrospinal Fluid Alteration	Sequelæ	Treatment
W. J., 20	G. S. W., left frontal	Lost	2·5 × 2·5 cm.	Right hemiparesis	Wounded 15 days before admission. No attempt at suture of scalp or débridement of wound	Protein, 65 mg. per cent 3 lymphocytes per c.mm.	Epilepsy (one fit)	Dressing. Chemotherapy
S. W., 30	Right temporal wound produced by bomb fragment	Not lost	2 × 2 cm.	Nil	Operation within a few hours, but wound suppurated	Protein, 55 mg. per cent 1 lymphocyte per c.mm.	Nil	Dressing. Chemotherapy
A. E., 59	Left frontal wound produced by bomb fragment	Lost	2 × 1 cm.	Nil	Early operation, but suppuration in wound	Not done	Nil	Dressing. Chemotherapy
L. H., 38	G. S. W., left parietal	?	2·5 × 1·5 cm.	Right hemiparesis and hemianæsthesia	Early treatment inadequate	Not done	Persisting cortical sensory loss on right	Dressing. Chemothearpy
G. B. P., 40	Buried in bombed building Left temporal compound depressed fracture	?	3 × 1 cm.	Right hemiparesis. Expressive aphasia	Operation 48 hours after injury. Wound suppurated	Protein, 50 mg. per cent White blood-corpuscles, 3 per c.mm.	Some residual dysphasia	Dressing. Chemotherapy
S. B., 19	Right temporal penetration produced by bomb fragment	Lost	2 × 1 cm.	Slight left hemiparesis	Inadequate débridement of small wound at primary operation	Protein, 100 mg. per cent Blood	Epilepsy during early stages. No recurrence	Dressing. Chemotherapy

* It will be noticed that in this group of cases the duration of the protrusion is not recorded. This is because of the absence of any tendency for projection of brain to occur through the defect after the first few days. Healing of the ulcer might, of course, be delayed for many weeks.

SUMMARY

1. The observations made in 11 cases of traumatic cerebral fungus are recorded.

2. It is pointed out that two types of fungus occur—the progressive and the non-progressive. The life history of a fungus is described and the clinical features of the condition are considered.

3. An account is given of the pathological anatomy of traumatic cerebral fungus.

4. The pathogenesis of cerebral fungus is discussed and the hypothesis is put forward that ventricular dilatation is the causative factor. This ventricular dilatation is itself produced by the alterations in intraventricular pressure which occur with each cardiac and respiratory cycle.

5. The treatment of the condition is discussed. The three essential features of treatment are the combating of infection of brain and meninges, the minimizing of injury to the exposed cerebral tissue, and the control of the tendency for progressive fungation to occur. The last is most readily accomplished by lumbar puncture.

6. The complications and sequelæ of cerebral fungus are briefly considered.

In conclusion, I must record my indebtedness to Miss Z. M. Stead for the care with which she has endeavoured to illustrate my conception of the pathological anatomy of cerebral fungus. It is also a pleasant duty to express my gratitude to Professor Paterson Ross for his interest and help in the preparation of the paper.

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TRACTION INJURIES TO THE LATERAL POPLITEAL NERVE AND TRACTION INJURIES TO PERIPHERAL NERVES AFTER SUTURE

BY W. BREMNER HIGHET AND W. HOLMES

NUFFIELD DEPARTMENT OF ORTHOPÆDIC SURGERY (PERIPHERAL NERVE INJURY CENTRE) AND DEPARTMENT OF ZOOLOGY, OXFORD

PLATT (1940) described 9 cases of traction injury to the lateral popliteal nerve. At the Oxford Centre we have observed 8 cases during the last two years: all have been subjected to operative exploration, and we have carried out detailed histological examination of the nerve lesions. In 3 of the cases an extensive resection was necessary before nerve suture was possible, and recovery failed to take place. We believe that the failure was due to a second traction injury inflicted on the nerve in the course of post-operative straightening of the knee, and that this injury was related to the extent of the original resection. There is no doubt that extensive lesions of peripheral nerves can be resected and the stumps sutured (Forrester-Brown, 1921; Babcock, 1927), but it seems that there is a limit to the extent of resection compatible with recovery. In an attempt to determine the maximum extent of resection compatible with recovery in other nerves and after other types of lesion, we have reviewed 42 other cases of nerve suture performed at the Oxford Centre.

TRACTION INJURIES TO THE LATERAL POPLITEAL NERVE

Platt has given a detailed description of this type of closed injury to the lateral popliteal nerve. The injury follows a severe adduction strain of the knee by which the lateral collateral ligament is ruptured and a subluxation or actual dislocation is produced. The tendon of the biceps may be ruptured, or there may be an avulsion of part of the head of the fibula. The iliotibial tract may be ruptured, and frequently the cruciate ligaments of the knee are damaged. The lateral popliteal nerve is subjected to severe traction. In some cases the nerve is completely ruptured, in others its macroscopical continuity is preserved; but in all our cases and in all but one of Platt's the injury was followed by complete paralysis.

CASE REPORTS

Case 1 (B. 20).—

HISTORY.—A farm labourer, born in 1918, joined the army in February, 1940. On Dec. 1 he was on duty in the cook-house carrying an armful of trays. He slipped and fell down a flight of eight stairs. His right leg twisted beneath him and he sustained a subluxation of the knee, with avulsion of the head of the fibula. He suffered great pain, but got up and tried to walk: the knee "went out like an elbow". There was an immediate complete lateral popliteal paralysis. On Dec. 12 an operation was performed at another hospital. The head of the fibula was replaced, the lateral popliteal nerve was stated to be "stretched, but intact".

On June 3, 1941, he was admitted to the Wingfield-Morris Orthopædic Hospital. Examination of the right knee showed considerable cruciate laxity, and obvious lateral instability from insufficiency of the lateral collateral ligament. The tendon of biceps and the iliotibial tract appeared intact. He showed complete paralysis of the lateral popliteal nerve.

FIRST OPERATION (June 18).—Resection and nerve suture (W. B. H.).

Anæsthesia: general, supplemented with local infiltration with 1–50,000 adrenaline in saline.

Approach.—Incision from lower border of gluteus maximus to upper third of leg. The lateral popliteal nerve was exposed throughout its length with relative

ease, and was stripped from the medial popliteal component of the sciatic within the limits of the exposure.

The Lesion (*Fig.* 209).—The sciatic bifurcation lay 10 cm. above the head of the fibula. Immediately below the bifurcation the lateral popliteal trunk was

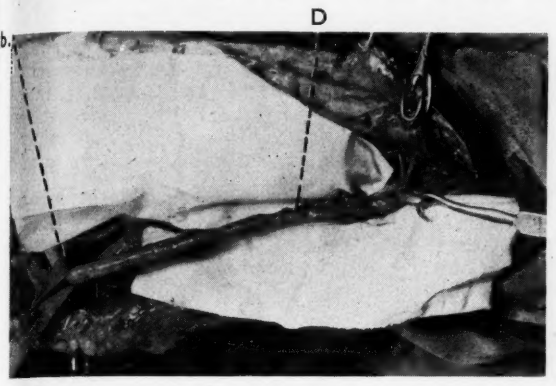

FIG. 209.—*Case* 1. Lateral popliteal nerve exposed at operation, June 18, 1941. The proximal end is to the left. S.b., Sciatic bifurcation; D, Distal level of resection.

6 mm. in diameter; 2 cm. below the bifurcation the nerve began to thicken gradually and became progressively firmer to palpation, until at 3 cm. above the head of the fibula it attained a diameter of 4 mm. × 7 mm. Distal to this point the nerve gradually decreased in diameter, until at 1 cm. below the head of the fibula its diameter was 5 mm. × 2 mm. At the level of the neck of the fibula the nerve became involved in scar tissue surrounding the origin of peroneus longus. The macroscopical appearance was not that of a complete division of the nerve, but for a length of more than 11 cm. the nerve was abnormally hard to palpation.

Faradic stimulation proved entirely negative.

Procedure.—Trial section of the thickest part of the lesion showed only scar tissue. Twelve serial sections were then made along the proximal trunk: nerve-bundles were not visible in the cross-section until 10 cm. above the lower end of the thickened segment. (This level, the proximal level of resection, was 2 cm. above the sciatic bifurcation and is not visible in *Fig.* 209.) Sections were then made distally and nerve-bundles were encountered 1·5 cm. below the distal end of the thickened segment. Accordingly, the total length of nerve resected amounted to 11·5 cm.

The nerve was mobilized from 5 cm. below the head of the fibula to beneath the gluteus maximus. The gain in length by mobilization amounted to 1·5 cm. The length gained by extension of the hip was 1 cm., and by flexion of the knee to 100°, 9 cm. The total length gained was thus 11·5 cm. The upper and lower portions of the incision were closed, and nerve suture was performed with six epineural sutures of fine black silk. With the knee flexed to 120° the suture line was not under tension, and lay 1·5 cm. above the head of the fibula.

POST-OPERATIVE TREATMENT.—A plaster spica was applied with the hip extended and the knee flexed to 120°. This plaster was changed after 23 days and a turnbuckle plaster applied (*Fig.* 210). The knee was gradually straightened by means of turnbuckles. By Sept. 9 (83 days after suture) the knee was fully extended and the plaster was removed.

HISTOLOGICAL REPORT.—The segments of nerve resected were fixed in formol-saline. Alternate pieces were embedded in paraffin wax, and the rest were first mordanted with 3 per cent aqueous potassium dichromate for 14 days at 37° C. before embedding. Transverse and longitudinal sections were cut: the

FIG. 210.—*Case* 2. Turnbuckle plaster applied July 30, 1941.

sections of the mordanted material were stained overnight in Kulschitzky's hæmatoxylin, differentiated by Pal's method and counterstained with alum carmine. In this way the myelin sheaths of myelinated fibres were demonstrated. The sections of the other material were stained with a trichrome stain or by silver methods for the demonstration of nerve axons, or with other stains for special tissue elements. Similar histological methods were used on all the material described in this series.

The nerve-fibres had undergone degeneration throughout the whole length of nerve resected. As a result of this degeneration the nerve-bundles were composed of neurilemmal or Schwann tubes containing proliferated Schwann cells (*see* Holmes and Young, 1942). The Schwann tubes contained

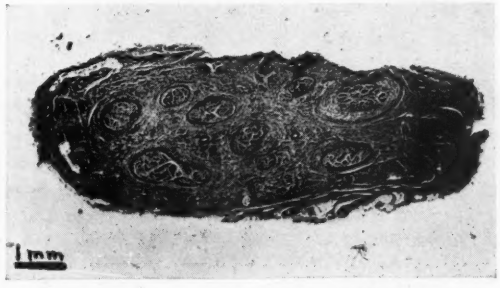

FIG. 211.—*Case* 1. Transverse section of the nerve at the distal end of the resected lesion, June 18, 1941.

regenerating fibres, but they were all of small diameter, and only a few were myelinated. Some of these fibres had succeeded in growing along the whole length of the nerve, but at the distal end of the lesion they were as few in number and as small in diameter as those which often succeed in reaching the distal stump of a nerve after a lesion producing

complete division. However, the nerve had not been completely divided at any point, for the Schwann tubes representing the original nerve-fibres passed all along its length. But at all levels the internal architecture of the nerve had been seriously disturbed.

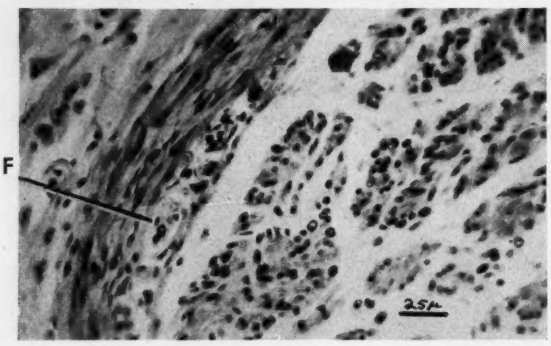

FIG. 212.—*Case* 1. Part of a nerve-bundle at the proximal end of the resected lesion. The nerve-fibres are all small in diameter, and at the edge of the bundle some of them (F) have been involved in fibrosis of the perineurium. (Weigert-carmine.)

Even at the proximal and distal ends of the lesion there was considerable dense collagenous thickening of the epineurium around and between the nerve-bundles (*Fig.* 211). And in *Fig.* 212 it may be seen that there had been fibroblastic proliferation in the perineurium of the bundle, and this has spread within

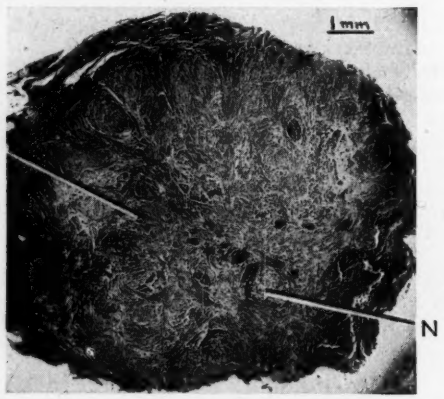

FIG. 213.—*Case* 1. Transverse section of the nerve in the middle of the lesion, June 18, 1941. The nerve-bundles are broken up by fibrosis, but nervous tissue can be identified throughout the section, e.g., as indicated (N).

the bundle so that some of the Schwann tubes, with their regenerating fibres (F), are surrounded by proliferated fibrous tissue.

The rest of the nerve resected showed still more marked abnormality, and at the level where the nerve was abnormally swollen, the nerve-bundles had been completely broken up by collagen formation, and the thickening of the trunk was due to this gross fibrosis (*Fig.* 213). The nervous tissue was represented chiefly by small groups of Schwann tubes separated from each other by the endoneural fibrosis, and in regions where the nerve showed some remains of its fascicular arrangement there was considerable œdema in the bundles and there were signs of further fibroblastic invasion.

Several of the arterial trunks within the damaged nerve showed considerable thickening of the intimal layer, with partial obliteration of the lumen of the vessel.

We concluded that although there was some continuity of nervous tissue throughout the lesion, the abnormality was so great that no worth-while spontaneous recovery could have been expected.

The condition of the sutured stumps, represented by the r roximal and distal ends of the excised segment, gave hope of recovery after suture. Nerve-bundles of reasonably normal appearance were present in both stumps, and the proximal stump contained regenerating fibres which could presumably grow out into the distal bundles.

Case 2 (M. 24).—

HISTORY.—On Sept. 26, 1940, the patient, an R.A.F. officer, born in 1908, was playing a game called 'high-cock-a-lorum' in Singapore: five people piled on top of him as he stood with his knees straight and his feet apart. The left knee gave way, and he sustained a subluxation with rupture of the lateral collateral ligament. He had excruciating pain for a few seconds only. There was an immediate complete lateral popliteal paralysis. A hæmarthrosis of the knee required aspiration.

June 30, 1941.—Admitted to the Wingfield-Morris Orthopædic Hospital. Examination showed considerable crucial laxity and some lateral instability of the left knee. The tendon of biceps and the iliotibial tract appeared intact. There was a complete lateral popliteal paralysis.

FIRST OPERATION (July 9, 1941).—Resection and suture of the lateral popliteal nerve (W. B. H.). Local anæsthesia (0·5 per cent planocaine with 1–50,000 adrenaline).

Approach.—As in *Case* 1.

State of Scar.—There was considerable scarring over the lateral aspect of the knee-joint involving the tendon of biceps, the lateral collateral ligament, the lateral head of gastrocnemius and the origin of

FIG. 214.—*Case* 2. The lesion exposed at operation, July 9, 1941. The proximal end is to the left. D, Distal level of resection; P, Proximal level of resection; S.b., Sciatic bifurcation; Sur., Sural communicating nerve.

peroneus longus and soleus. The intermuscular fascia of the thigh was discoloured from old hæmorrhage.

The Nerve Lesion (*Fig.* 214).—The sciatic bifurcation lay 11 cm. above the head of the fibula. At this level the diameter of the lateral popliteal nerve was 7 mm. Immediately below the bifurcation the nerve expanded to form an obvious neuroma 25 mm. in

length and of a maximum diameter of 14 mm. × 10 mm. The neuroma was smooth but densely hard, and the proximal trunk was indurated for at least 2 cm. abbve it. Distal to the neuroma the nerve-trunk narrowed to a diameter of 4 mm., and after a

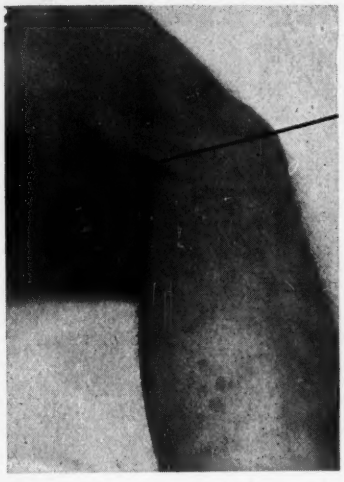

FIG. 215.—*Case* 2. Outer aspect of the knee, Nov. 7, 1941, showing the taut lateral popliteal nerve, L.p.

course of 1 cm. divided into the main trunk and the sural communicating nerve (ramus communicans fibularis). The two divisions were separated proximally as far as the lower margin of the neuroma. The diameter of the lateral popliteal nerve itself immediately below the neuroma was 2 mm. only. After a course of 4 cm. the nerve became involved in dense scar tissue; 1 cm. below the head of the fibula the nerve-trunk presented a slight swelling and was 9 mm. × 6 mm. in diameter. Thereafter the nerve passed normally around the neck of the fibula and broke up into its various branches. At the

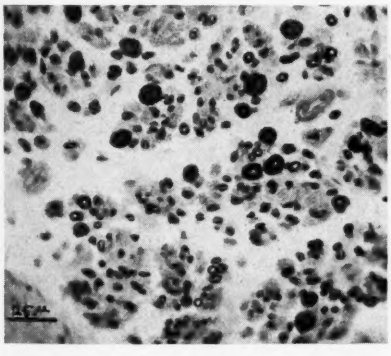

FIG. 217.—*Case* 2. Part of a nerve-bundle at the proximal end of the lesion, July 9, 1941. Many myelinated fibres are present, and some of them are of normal diameter. (Weigert-carmine.)

evel of the neck of the fibula the nerve was normal to palpation and its diameter 6 mm. × 3 mm. Thus the macroscopical appearance was that of a complete division of the nerve-trunk, the proximal and distal stumps being connected only by a narrow strand of scar tissue.

Results of stimulation: Motor response nil. A sensory response was obtained on strong faradic stimulation as far distally as 7 cm. below the lower end of the neuroma, but below this was negative. The sural communicating nerve gave a positive sensory response.

Procedure.—Two stay sutures were inserted at the proximal and distal ends of the lesion, 16·5 cm. apart. A gain in length of 2·5 cm. was obtained by mobilization of the nerve from the neck of the fibula to several centimetres above the lower margin of gluteus maximus. Flexion of the knee to 90° afforded a gain of an additional 6 cm., flexion of the knee from 90° to 130° gave an additional 5·5 cm., extension of the hip gave a further gain of 1 cm. Thus the

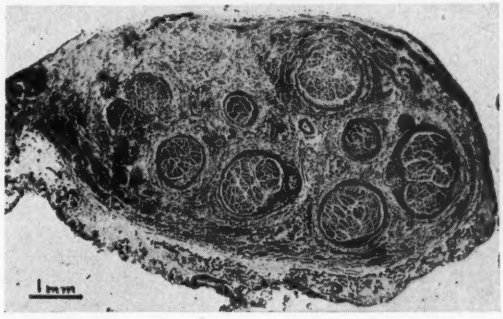

FIG. 216.—*Case* 2. Transverse section at the proximal end of the resected lesion, July 9, 1941.

total gain in length was 15 cm. Resection was performed until normal nerve-bundles were reached. This involved section 2·5 cm. above the proximal end of the neuroma, and 0·5 cm. below the distal swelling. The total length resected was 15 cm. Suture was performed with six epineural sutures of fine black silk. The suture line was not under tension, and lay 1·5 cm. below the head of the fibula.

POST-OPERATIVE TREATMENT.—A plaster spica was applied with the hip extended and the knee flexed to 130°. This plaster was changed after 21 days and a turnbuckle plaster applied (*see Fig.* 210). The knee

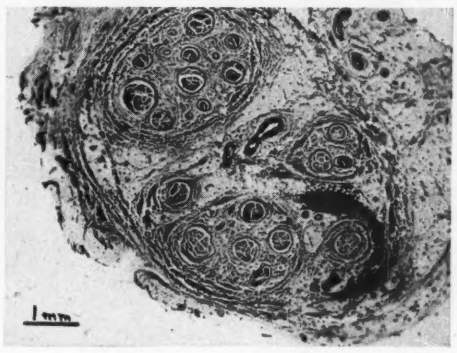

FIG. 218.—*Case* 2. Transverse section at the distal end of the resected lesion, July 9, 1941.

was gradually straightened by means of turnbuckles. On Sept. 6 the spica was replaced by a plaster extending from the toes to the groin, and straightening of the knee was continued. At this time it was noted that the lateral popliteal nerve was palpable as a taut cord medial to the tendon of biceps (*Fig.* 215). The plaster was removed on Sept. 22 when the knee had been extended to 45° short of full extension. He was then allowed to walk. He obtained full extension of the knee by Nov. 7 (121 days after nerve suture).

HISTOLOGICAL REPORT.—The nerve had undergone complete rupture. The proximal stump terminated in a bulbous neuroma: from this some fibres had grown out into the strand of scar tissue, but very few of them had reached the distal stump.

At the proximal end of the resected lesion the nerve-bundles were separated by some interfascicular epineural fibrosis, but this was not severe (*Fig.* 216), though in one or two bundles the perineurium also was thickened by fibrosis. Many myelinated nerve-fibres were present in the nerve at this level, but they were of less than normal diameter (*Fig.* 217). At the distal end of the lesion, representing the distal stump sutured, there was some interfascicular fibrosis, but the fibrous tissue was not dense (*Fig.* 218). There was intimal thickening in the walls of several arteries in the nerve, in some cases with almost complete obliteration of the lumen.

As the proximal stump sutured contained many myelinated nerve-fibres, and intact bundles were present in the distal stump, it appeared that a good recovery might be expected after suture.

Case 3 (R. 6).—An army private born in 1918.

HISTORY.—On Dec. 11, 1940, he was tackled while playing rugby football and sustained an adduction strain of the right knee with rupture of the lateral collateral ligament. There was an immediate complete lateral popliteal paralysis. He was admitted to hospital elsewhere, and 18 days after the injury the right medial semilunar cartilage was excised (no note of operative findings).

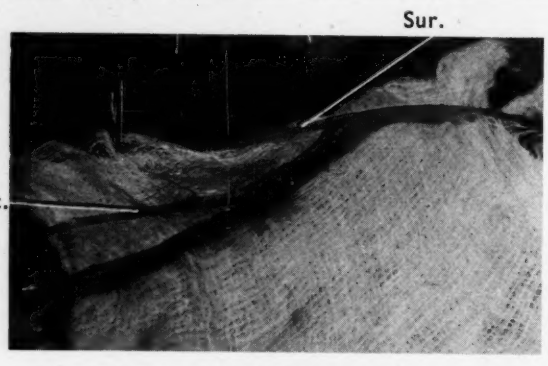

FIG. 219.—*Case* 3. The lesion exposed at operation, April 1, 1941. The proximal end is to the right. Sur., Sural communicating nerve; L.c., Lateral cutaneous nerve of the leg.

On March 23, 1941, he was admitted to the Wingfield-Morris Orthopædic Hospital. Examination showed an obvious rupture of the lateral collateral ligament of the right knee, with lateral instability and cruciate laxity. The tendon of biceps and the iliotibial tract appeared intact. There was a complete lateral popliteal paralysis.

OPERATION (April 1).—Exploration and suture of lateral popliteal nerve (W. B. H.).

The operation was commenced under local, and completed under general, anæsthesia. There was dense scarring over the lateral aspect of the knee.

The Nerve Lesion (*Fig.* 219).—The sciatic bifurcation lay 15 cm. above the head of the fibula. At this level the diameter of the lateral popliteal nerve was 5 mm. There was an obvious proximal neuroma—length 10 mm., diameter 8 mm.—lying 6 cm. below the sciatic bifurcation. Below the neuroma for a distance of 4 cm. the nerve-trunk was encased in dense scar tissue beneath the medial border of the tendon of biceps. The diameter of the distal trunk at the level of the head of the fibula was 5 mm. × 3 mm. When the distal trunk was traced upward it was found to wither gradually to a diameter of 3 mm. at the lower limit of the zone of scarring. The macroscopical appearance of the lesion was intermediate between that in *Cases* 1 and 2. There was no obvious complete division, but over a length of 11 cm. the nerve was grossly abnormal.

Results of faradic stimulation: No motor response. A sensory response was obtained on stimulation of the whole of the distal trunk, but a strong stimulus was required.

Procedure.—Eleven centimetres of the nerve-trunk were resected. The proximal section lay 3 cm. above the lower end of the neuroma, and the distal section 4 cm. below the zone of scarring (*Fig.* 219). The lateral cutaneous nerve of the leg arising from the mid-portion of the zone of scarring was sacrificed; the sural communicating nerve arising above the neuroma was preserved. Suture was performed with six black silk epineural sutures. The site of suture lay 1 cm. above the head of the fibula.

POST-OPERATIVE TREATMENT.—Stretching was carried out in a manner similar to that in the previous cases, and the knee was fully extended 58 days after operation.

HISTOLOGICAL REPORT.—The nerve-fibres had degenerated throughout the length of nerve resected. A large number of regenerating axons had succeeded in crossing the lesion, but even at its proximal end they were all of small diameter and only few of them were myelinated (*Fig.* 220).

At all levels in the lesion recognizable nervous tissue was present in the form of re-innervated Schwann tubes, often collected into bundles, but at the level of the proximal swelling on the nerve there were signs that some of the tubes had undergone rupture, and that the swelling was partly due to the outgrowth of Schwann cells, fibroblasts, and regenerating axons from their ends. However, many of the bundles preserved their continuity, so the nerve had not been completely interrupted at any point. Throughout the lesion, and particularly at the level of the proximal swelling, there had been considerable fibrosis of the nerve-sheaths around and between the bundles, and proliferation of fibroblasts within the bundles themselves. This fibrosis was present, though not at its worst, even at the proximal and distal ends of the resected nerve (*Fig.* 221, A, B). At

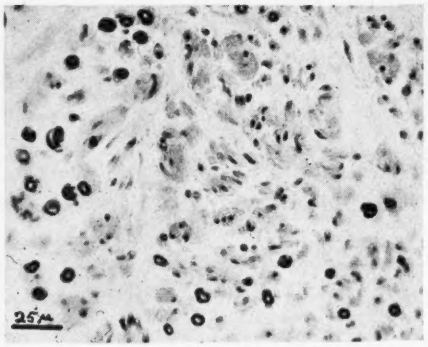

FIG. 220.—*Case* 3. Transverse section of part of a nerve-bundle from the proximal end of the resected lesion, April 1, 1941. The nerve is composed of Schwann tubes of degeneration, in some of which regenerating myelinated fibres of various sizes are present. (Weigert-carmine.)

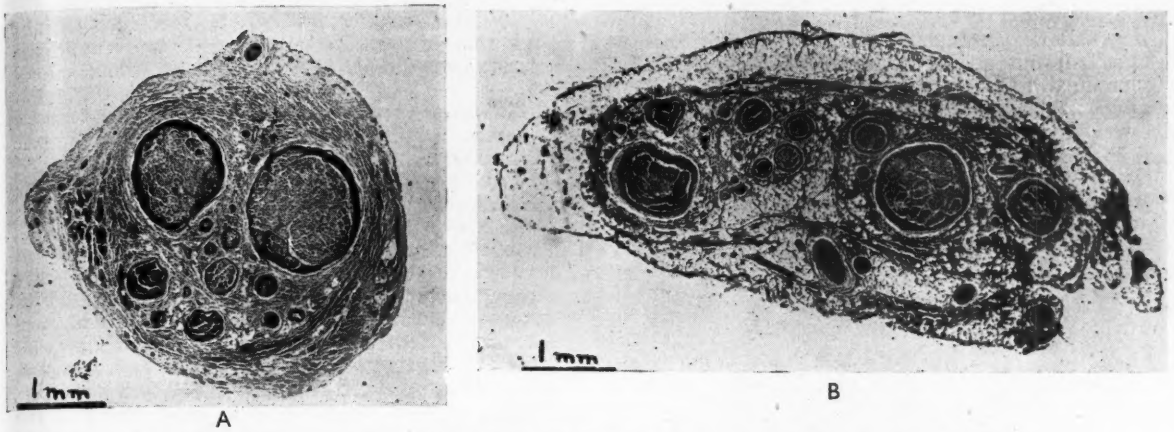

FIG. 221.—*Case* 3. A, Transverse section of the proximal end of the resected lesion, April 1, 1941. B, Transverse section of the distal end of the lesion, April 1, 1941.

the distal end the fibrous tissue was œdematous and less dense (*Fig.* 221, B). The arterial trunks at the distal end of the lesion showed some intimal thickening. As nerve-fibres were present in the proximal stump, and both stumps contained apparently undamaged nerve-bundles, we considered that recovery might be expected after suture.

Case 4 (M. 22).—A bricklayer born in 1900.

HISTORY (Jan. 29, 1941).—He fell from his bicycle and sustained a subluxation of the left knee with rupture of the lateral collateral ligament and avulsion of the tendon of biceps femoris. There was an immediate complete lateral popliteal paralysis.

On March 4 operation was performed at another hospital. The lateral semilunar cartilage was removed

FIG. 222.—*Case* 4. The lesion exposed at operation, Sept. 24, 1041. The proximal end is to the left.

because of a marginal tear. The avulsed tendon of biceps and the lateral collateral ligament were repaired. The lateral popliteal nerve was inspected and stated to be "partly, but probably not completely ruptured".

On Sept. 22 admitted to Wingfield-Morris Orthopædic Hospital. Examination showed lateral instability and cruciate laxity of the knee. There was a complete lateral popliteal paralysis.

OPERATION (Sept. 24).—Exploration and suture of the lateral popliteal nerve (W. B. H.). General anæsthesia. There was extremely dense scarring of the postero-lateral aspect of the knee.

The Nerve Lesion (*Fig.* 222).—The lesion was less extensive than in *Cases* 1, 2, and 3. At the level of the knee-joint there was a proximal neuroma 10 mm. in diameter. Below this there was a narrow band of scar tissue 3 cm. in length. Nervous tissue was not apparent below this until the level of the neck of the fibula, at the site of bifurcation of the nerve into musculo-cutaneous and anterior tibial divisions. The macroscopical appearance was thus that of complete division of the nerve, continuity being maintained only by the band of scar tissue.

Results of stimulation: Negative.

Procedure.—It was necessary to resect only 6 cm. of nerve to obtain stumps which in cross-section had a macroscopically satisfactory appearance. The distal level of resection of the nerve lay below the bifurcation. Suture was performed without difficulty at the neck of the fibula.

POST-OPERATIVE TREATMENT.—Stretching was performed as in the other cases by means of a turnbuckle plaster. The knee was fully extended by Nov. 25 (62 days after nerve suture).

HISTOLOGICAL REPORT.—The nerve had been

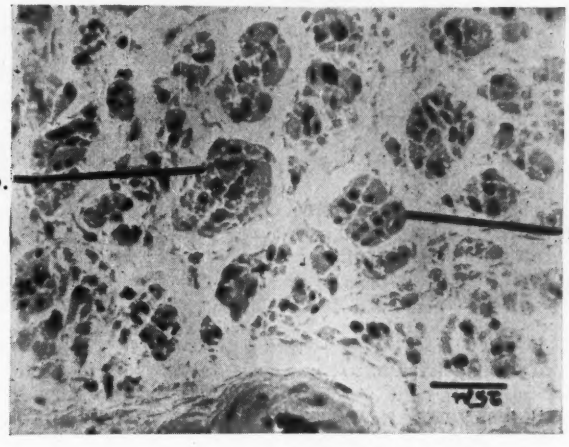

FIG. 223.—*Case* 4.—Transverse section of part of a nerve-bundle at the proximal end of the lesion, Sept. 24, 1941. The nerve is composed of Schwann tubes of degeneration (S.t.) containing a very few small regenerating myelinated fibres. (Weigert-carmine.)

completely interrupted, and its proximal stump was connected to the distal stumps by a band of scar tissue containing no nervous elements. The nerve-fibres in

the proximal stump had undergone retrograde degeneration throughout the length of it that was resected, but at the proximal level of resection it contained a few small regenerating myelinated fibres (*Fig.* 223). At this level the nerve-bundles were

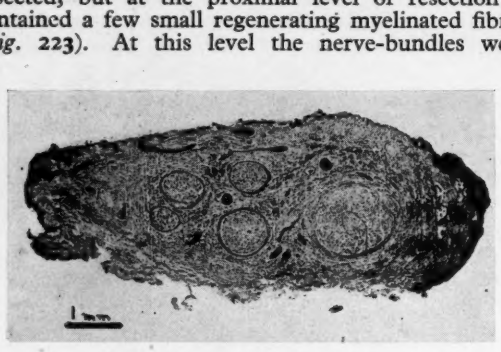

FIG. 224.—*Case* 4. Transverse section of the proximal end of the lesion, Sept. 24, 1941.

separated by dense fibrous tissue, and the perineurium of the bundles had undergone collagenous thickening (*Fig.* 224). The lumen of many of the arteries in the nerve was seriously reduced by intimal proliferation. The two distal stumps were not excessively fibrosed, and we considered that the condition of the sutured stumps was compatible with recovery.

Case 5 (A. 15).—An army private born in 1918.

HISTORY (June 4, 1941).—He met with a motor-cycle accident and sustained a fracture of the right femur and a dislocation of the left knee. There was an immediate complete left lateral popliteal paralysis.

On Sept. 30 he was admitted to Wingfield-Morris Orthopædic Hospital. Examination showed sound union of the right femur, but with 7 cm. of shortening. On the left side there was considerable cruciate laxity and lateral instability of the knee. The tendon of biceps and the iliotibial tract appeared intact. He presented a complete lateral popliteal paralysis.

FIRST OPERATION (Oct. 8).—Exploration and neurolysis of the lateral popliteal nerve (W.B.H.). General anæsthesia, supplemented by local infiltration.

State of Scar.—There was a mass of scar tissue over the postero-lateral aspect of the knee, extending for 10 cm. above the head of the fibula. The tendon of biceps was ruptured, but the torn ends were connected by a band of scar tissue.

The Nerve Lesion (*Fig.* 225).—Over a distance of 8 cm.—from 2 cm. to 10 cm. above the head of the fibula—the nerve was embedded in scar tissue, but was freed with relative ease. Over this length the nerve was compressed and thin, firmer than normal, but not hard. At its narrowest point (4 cm. above the head of the fibula) it was 4 mm. × 2 mm. in diameter.

Results of stimulation: Bipolar faradic stimulation —no motor response.

Procedure.—There seemed to be obvious continuity of nervous tissue, so neurolysis only was carried out.

The patient was 6 ft. 4 in. in height, and his disability was aggravated by the shortening of the right femur.

SECOND OPERATION (April 22, 1942).—Bone shortening of the left femur was performed by Professor Seddon; 7 cm. of the femur were excised, and fixation was secured by a 5-in. vitallium plate.

July 29: There was no evidence of recovery in the lateral popliteal nerve, and a further exploration was carried out. It was considered that owing to the shortening of the femur resection and nerve suture should be possible.

THIRD OPERATION (W. B. H.).—Eleven months after the original injury. There was very dense scarring over the whole field of the previous exploration.

Stimulation: There was no response in any muscle of lateral popliteal innervation on bipolar faradic stimulation of the nerve.

Procedure.—A guide suture was inserted 11 cm. above the head of the fibula. Section of the nerve 1 cm. above this revealed only a few nerve-bundles, obviously densely fibrosed, and the section was 'gritty'. A distal section 6 cm. below the guide suture showed only a few nerve-bundles amongst dense scar tissue. Thus the first resection amounted to 7 cm. With the knee flexed as far as possible (only 45°) the stumps could only just be approximated, and it was considered that, in view of their condition, suture was not worth while. Accordingly a full resection of the lesion was performed. The proximal stump was sectioned progressively at 2-cm. intervals. Normal nerve-bundles were not seen until a level 16 cm. above the head of the fibula. Similar sections were made distally, and normal bundles were not seen until the level of the head of the fibula was reached. The sural communicating nerve was stripped from the lateral popliteal trunk and left intact. The total length of the lesion resected thus amounted to 20 cm.

HISTOLOGICAL REPORT.—The nerve-fibres had degenerated throughout the whole 20 cm. length of nerve resected. A large number of regenerating axons had grown through the lesion, but at all levels they were all of very small diameter and few of them were myelinated. Nerve-bundles were continuous along the whole length of the lesion, and there had thus been no rupture of the nerve, and probably no rupture of the Schwann tubes. Throughout the lesion, and even at its extremities, the nerve-bundles were separated by masses of dense collagen and some œdematous tissue, and the nerve-trunk and the bundles were flattened and distorted, probably as a result of

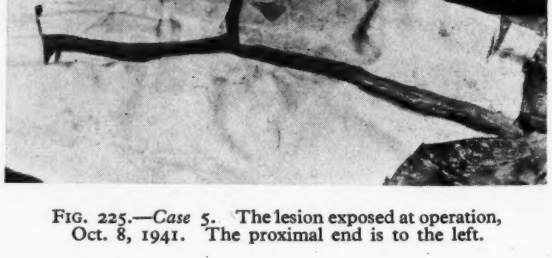

FIG. 225.—*Case* 5. The lesion exposed at operation, Oct. 8, 1941. The proximal end is to the left.

constriction by scar tissue. Many of the arteries in the trunk showed intimal thickening, and there were patches of lymphocytes around vessels. Within the nerve-bundles there was some endoneural œdema, and the perineurium was thickened by fibrosis.

Case 6 (H. 12).—An army private born in 1919.

HISTORY (Nov. 27, 1940).—Motor-cycle accident after twenty-first birthday party. He sustained a fracture of the right tibia and fibula and a subluxation of the left knee, with rupture of the lateral collateral ligament and a fracture of the tibial spine. There was an immediate complete lateral popliteal paralysis.

On Jan. 7, 1941, he was admitted to Wingfield-Morris Orthopædic Hospital. He showed signs of complete paralysis of the left lateral popliteal nerve.

FIRST OPERATION (March 10).—Repair of the tendon of biceps femoris; suture of end-bulbs of the lateral popliteal nerve (Professor Seddon). General anæsthesia.

Extent of Damage.—There were irregular patches of scar tissue subcutaneously over the lateral side of the knee, and it looked as if everything but the skin had been violently disrupted. The iliotibial tract had been ruptured and was replaced by scar tissue. The tendon of biceps had been torn at its lower end and become attached to the femoral condyle, just lateral to the posterior articular surface. The lateral collateral ligament was extremely lax, but the tear was repaired by fibrous tissue. There was extensive dirty-brown staining from old hæmorrhage.

The Nerve Lesion.—The upper end of the nerve was found torn across at a point 6 cm. above the knee-joint. There was no proximal neuroma. Over a distance of 5 cm. the nerve was irregularly firm and the sheath greatly thickened. After laborious dissection the lower end was found curled up and adherent to the femoral condyle anterior to the attachment of the lateral collateral ligament. There was no distal swelling, but the nerve was irregularly firm and the sheath thickened over a distance of 5 cm., just as was the proximal nerve-stump. After the stumps had been dissected free there was a total gap of 13 cm. between possibly healthy nerve. Trial sections 3 cm. from the extremities of the proximal and distal stumps showed only dense fibrous tissue. Immediate resection and suture was impossible since the flexion of the knee amounted to 85° only and the length gained amounted to 6·5 cm. only.

Procedure.—Mobilization of the nerve from the neck of the fibula to the lower border of gluteus maximus. Bulb suture was performed, the nerve-ends being overlapped for 3 cm. and sutured with three loops of silk. The centre of the bulb suture lay 7 cm. above the head of the fibula. The tendon of biceps was repaired.

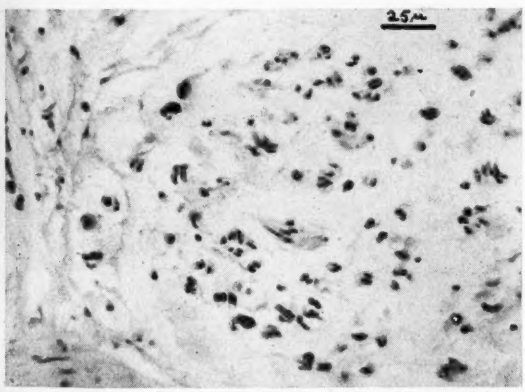

FIG. 227.—*Case* 6. Transverse section of part of a nerve-bundle from the proximal end of the lesion, Sept. 17, 1941. The Schwann tubes, containing small myelinated fibres, are separated by interstitial œdema. (Weigert-carmine.)

A turnbuckle plaster was applied with the hip extended and the knee flexed to 85°. The knee was extended over a period of five weeks and the plaster then removed.

The second stage operation was delayed because of attempts to improve the range of movement of the knee. However, by Sept. 17, 1941, the range of movement amounted to only 80° flexion.

SECOND OPERATION (Sept. 17).—Second-stage suture (Professor Seddon). General anæsthesia.

The site of the bulb suture was identified: its centre lay 9 cm. above the head of the fibula—that is, it had shifted proximally some 2 cm. during extension of the leg.

State of the Nerve.—The nerve appeared healthy down to a point 27 cm. above the head of the fibula. Below this level it became increasingly firm and the sheath thickened. Below the bulb suture the nerve was firmer for a few centimetres, but for 5 cm. above the head of the fibula it felt normal.

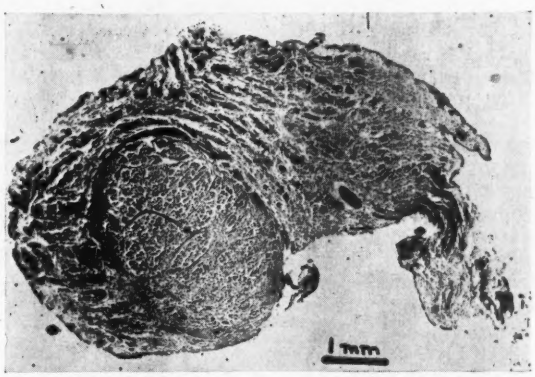

FIG. 226.—*Case* 6. Transverse section of the proximal end of the lesion, Sept. 17, 1941. The trunk contains one large nerve-bundle; the rest is fibrous tissue.

Procedure.—Several trial sections were made before healthy bundles were exposed. The total length resected amounted to 8·8 cm., the lower section lying 6 cm. above the head of the fibula. The whole sciatic trunk was mobilized as far as the lower border of the gluteus maximus; this gave a gain

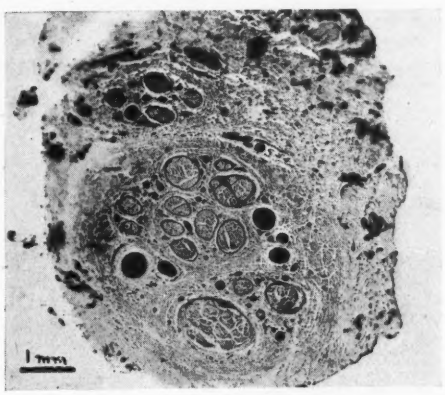

FIG. 228.—*Case* 6. Transverse section of the distal end of the lesion, Sept. 17, 1941.

of only 1 cm. Flexion of the knee to 80°—the maximum flexion obtainable—yielded a further 6·5 cm. Full extension of the hip yielded a further 2 cm. The total gain in length was thus 9·5 cm. The stumps were approximated with four epineural silk sutures and the suture completed with plasma (Seddon and Medawar, 1942). The suture line lay 6 cm. above the head of the fibula. Plaster was applied with the hip extended and the knee flexed to 80°. The plaster was left intact until Oct. 7 (20 days), and a turnbuckle

plaster was then applied. The knee was gradually straightened and the plaster was finally removed on Dec. 5, when the knee was extended to 20° short of full extension.

HISTOLOGICAL REPORT.—At the site of bulb suture the proximal and distal nerve-trunks were swollen by a massive proliferation of collagen. This was laid down in thick strands orientated along the long axis of the nerve, and it was present around and between the nerve-bundles. The suture material used at the operation of bulb suture could be identified in the sections, and it was clear that a thick layer of collagen had been deposited around the nerve after the suture. The arteries in the nerve showed intimal thickening, and there were some lymphoid aggregates in the neighbourhood of blood-vessels.

At the proximal end of the resected nerve—that is, at the proximal surface sutured at the second operation—nerve-bundles were visible (*Fig.* 226), but they showed considerable abnormality. The Schwann tubes, which contained some regenerating myelinated fibres, were separated from each other by considerable œdema spaces (*Fig.* 227), and in places at the edge of the bundle fibroblasts had laid down strands of collagen in the œdematous endoneural tissue. At the distal end of the lesion the nerve-bundles were separated by a dense proliferation of fibrous tissue (*Fig.* 228).

We were not able to decide whether the œdema in the proximal stump and the fibrotic changes resulted from the original traction lesion or from the process of stretching after bulb suture; nor could we say from the histological evidence whether recovery was likely to take place after suture, as we did not know what hindrance to regeneration might arise from the œdematous condition of the proximal stump.

Case 7 (K. 7).—A Czech soldier born in 1907.

HISTORY.—On Feb. 16, 1942, he slipped and fell when jumping a ditch; he did not know in what position he fell. There was an immediate complete lateral popliteal paralysis. A hæmarthrosis of the knee required aspiration.

FIG. 229.—*Case* 7. The lesion exposed at operation, July 15, 1942.

On April 7 he was admitted to Wingfield-Morris Orthopædic Hospital. There was slight limitation of movement in the knee—flexion 20°–120°. There was slight cruciate laxity, but no lateral instability. The tendon of biceps and the iliotibial tract appeared intact. There was a complete lateral popliteal paralysis. The nerve could be palpated for several centimetres above the head of the fibula; it was firmer than on the normal side, and squeezing caused no tingling sensation as it did on the normal side.

A radiograph of the right knee showed no abnormality.

July 15: No evidence of motor or sensory recovery.

OPERATION (July 15).—Exploration and suture of the lateral popliteal nerve (W. B. H.).

Local anæsthesia: 0·5 per cent procaine with 1–200,000 adrenaline.

State of Scar.—There was moderate scarring of all soft tissues over the lateral aspect of the knee extending as high as the mid-thigh. There was considerable scarring around the lateral collateral ligament.

The Nerve Lesion (*Figs.* 229, 230).—There was a large fusiform neuroma centred 14 cm. above the head of the fibula and just below the sciatic bifurcation. The diameter of the normal proximal and distal trunks was 5 mm., and the maximum diameter of the neuroma 13 mm. The neuroma was densely hard and the nerve-trunks were thickened and indurated for 6 cm. above and 7 cm. below the centre of the neuroma. The lateral cutaneous nerve of the leg arose 2 cm. above the head of the fibula. The sural communicating nerve arose 6 cm. below the centre of the neurmoa, but appeared normal. It was stripped upwards from the lateral popliteal trunk as far as 20 cm. above the head of the fibula, and though adherent to the medial side of the neuroma, it appeared normal over its whole course. The medial popliteal nerve also appeared normal.

Stimulation: Faradic stimulation gave no motor response. The distal trunk and the lateral cutaneous nerve of the leg gave a sensory response even at a level 2 cm. below the head of the fibula (tingling over the dorsum of the foot), but a strong stimulus was required. By running the electrodes up the main nerve-trunk we found that a response to a minimal stimulus was given only at 8 cm. above the centre of the lesion. The medial popliteal and sural nerves gave a sensory response to minimal stimulation over their whole length. We concluded that there were some sensory fibres in the distal trunk, but that they must be few or abnormal as so large a stimulus was necessary to elicit a response. Moreover, the results of stimulation showed that the nerve was abnormal as high as 8 cm. above the level of the lesion. Stimulation of the distal trunk after resection of the lesion was entirely negative.

Procedure.—Trial section of the centre of the neuroma (*Fig.* 230) revealed only dense scar tissue

FIG. 230.—*Case* 7. The fusiform neuroma after resection. The trial section made at operation can be seen in the middle of the specimen.

containing possibly a few tiny nerve-bundles. From our experience in the previous cases it seemed probable that a resection of at least 15 cm. would have been necessary to obtain stumps of a normal macroscopical appearance, and it was decided in this case to perform only a limited resection. Accordingly, 7 cm. of the lesion was resected—3·5 cm. above and below the middle of the neuroma. The appearance of the stumps after this resection was macroscopically poor, as there was considerable intraneural scarring, though some nerve-bundles were visible. Suture was performed with seven epineural silk sutures after mobilization of the nerve from the neck of the fibula to the lower border of gluteus maximus, and flexion of the knee to 110°.

Post-operative stretching was carried out as in the previous cases.

HISTOLOGICAL REPORT.—The lesion was a typical fusiform neuroma—that is to say, the nerve had retained complete continuity, but was swollen to more than twice its normal diameter in the middle of the lesion. The specimen was examined in transverse section at its proximal and distal ends and in median longitudinal section.

Although the nerve was macroscopically continuous, the nervous elements within it had undergone complete rupture. Thus the lesion differed from a complete division only in so far as the epineural sheath had remained intact, thus preserving the continuity of the trunk (*Fig.* 231). A few nerve-bundles were visible at the proximal and distal ends of the lesion (*Fig.* 231), but the neuroma was chiefly composed of an irregular mass of collagen at some points laid down as thick and relatively acellular masses and at others deposited as thin strands. Much of this collagen appeared to have been formed by fibroblasts which had grown out from the proximal and distal stumps

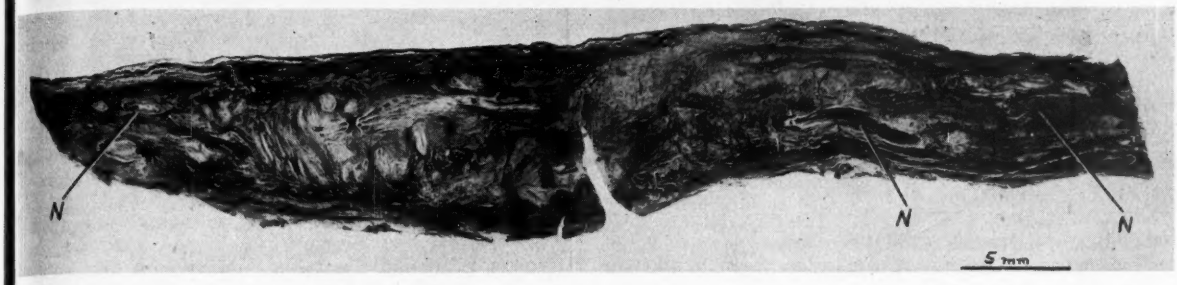

FIG. 231.—*Case* 7. A longitudinal section of the fusiform neuroma. A few nerve-bundles (N) are visible at the proximal and distal ends of the lesion.

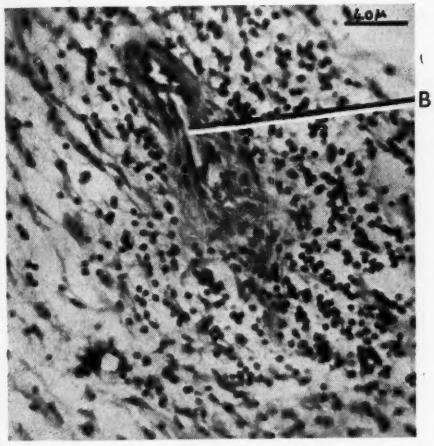

FIG. 232.—*Case* 7. A mass of lymphocytes around a blood-vessel (B) in the neuroma. (Masson.)

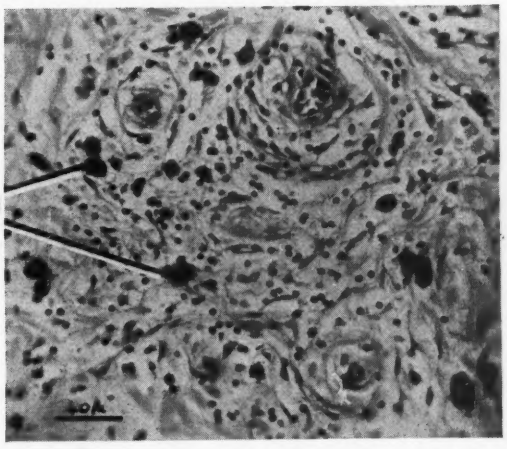

FIG. 233.—*Case* 7. Lymphocytes and macrophages containing iron pigment (M) around small blood-vessels in the neuroma. (Prussian-blue method, basic fuchsin.)

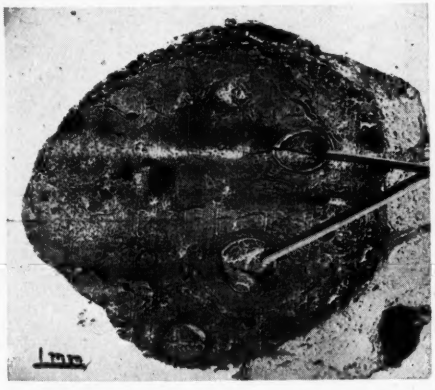

FIG. 234.—*Case* 7. Transverse section of the proximal end of the lesion. The nerve-bundles (N) are separated by dense fibrous tissue.

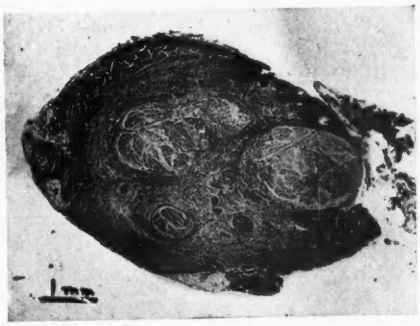

FIG. 235.—*Case* 7. Transverse section of the distal end of the lesion.

of the ruptured nerve-bundles; and the neuroma also contained columns of Schwann cells which had grown out similarly, and axons regenerating from the proximal stump. Many of the arteries showed severe intimal thickening throughout the lesion. Scattered throughout the neuroma were lymphoid aggregates, in the neighbourhood of blood-vessels (*Fig.* 232), and there were many foam cells whose cytoplasm was loaded with material which gave the Prussian-blue reaction with potassium ferrocyanide—presumably degraded hæmoglobin (*Fig.* 233).

The nerve-fibres had undergone degeneration throughout the resected segment. The proximal bundles contained many regenerating axons, and many of these had reached the distal stump, but in both proximal and distal stumps the axons were of small diameter and very few of them were myelinated. At the proximal level of resection some nerve-bundles were visible (*Fig.* 234), but there was dense interfascicular fibrosis, and some of the bundles were broken up by endoneural collagenization. A similar condition existed at the distal end of the lesion where there was also endoneural œdema (*Fig.* 235).

Case 8 (T. 24).—An Army private born in 1915.

HISTORY (Jan. 26, 1942).—While riding a motor-cycle he collided with an oncoming lorry. He

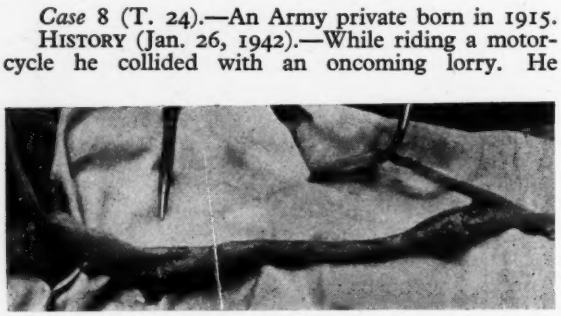

FIG. 236.—*Case* 8. The lesion exposed at operation, Aug. 12, 1942. The proximal end is to the right.

remembers no details of the accident. He was admitted to hospital elsewhere with multiple injuries, including a subluxation of the right knee with rupture

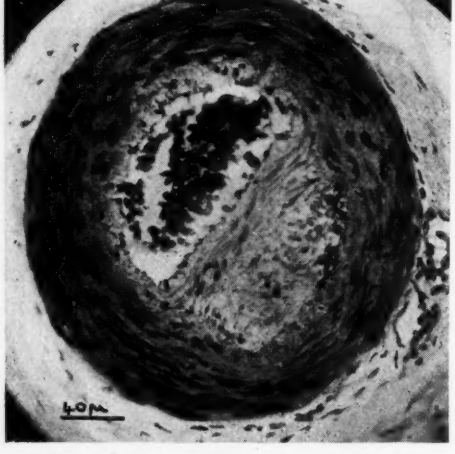

FIG. 237.—*Case* 8. Endarteritis in one of the vessels of the nerve. (Masson.)

of the lateral collateral ligament, avulsion of the tendon of biceps, and rupture of the cruciate ligaments. There was also severe circulatory disturbance and the popliteal, posterior tibial, and dorsalis pedis pulses were not palpable. A plaster back splint was applied. The early notes are meagre, but on April 15 there was said to be moist ulceration over the dorsum of the base of the first metatarsal, along the plantar surface of the fifth metatarsal, and over the centre of the sole of the foot. This was thought to indicate gangrene. Amputation was considered, but rejected in favour of conservative treatment. The areas of ulceration were treated with sulphapyridine powder, and the leg was elevated. By May 25 all areas were healed and dry.

On July 31 he was admitted to Wingfield-Morris Orthopædic Hospital. In the right knee there was no swelling or synovial effusion, and there was a full range of passive movement. There was marked cruciate laxity and lateral instability due to rupture of the lateral collateral ligament. There was an obvious rupture of the tendon of biceps, and X-ray examination showed avulsion of a fragment of the head of the fibula.

The popliteal, posterior tibial, and dorsalis pedis pulses were not palpable. Oscillometry showed negligible pulsations below the knee. There was Volkmann's ischæmic necrosis of the whole anterior tibial group of muscles and the peronei.

There was an incomplete lesion of the medial popliteal nerve, probably secondary to the circulatory disturbance. There was a complete lateral popliteal paralysis (motor, sensory, and sweating).

OPERATION.—(Aug. 12).—Operative exploration (W. B. H.). General anæsthesia. An arteriograph was taken and showed obstruction of the popliteal artery in the popliteal space, and on biopsy the lateral popliteal group of muscles showed obvious Volkmann's ischæmic necrosis. There was a rupture of the lateral collateral ligament and an avulsion of the tendon of biceps. The tendon of biceps was later repaired.

The Nerve Lesion.—The lesion of the lateral popliteal nerve was two-fold (*Fig.* 236): (*a*) In the region of the head of the fibula where a fragment of bone had been avulsed with the tendon of biceps, the nerve was caught up in scar tissue, angulated forwards,

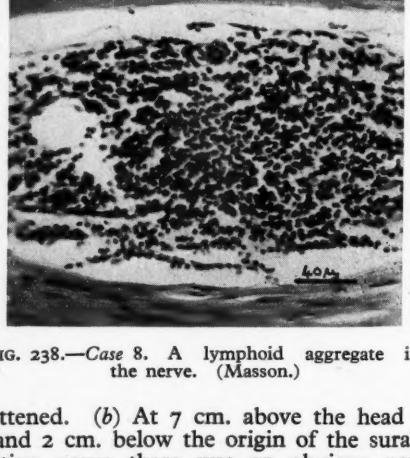

FIG. 238.—*Case* 8. A lymphoid aggregate in the nerve. (Masson.)

and flattened. (*b*) At 7 cm. above the head of the fibula and 2 cm. below the origin of the sural communicating nerve there was an obvious neuroma, 22 mm. in length, 10 mm. in maximum diameter, and of very hard consistency. Below the neuroma was a thin strand of indurated tissue which appeared to consist entirely of scar tissue. Above the neuroma the nerve-trunk was extremely indurated for at least

10 cm. Trial sections were made at various levels, but no nerve-bundles were visible.

There seemed to be no possibility of nerve suture; moreover, on account of the ischæmic necrosis of the muscles supplied by the lateral popliteal nerve, it was considered that regeneration in the nerve could be of no benefit to the patient. Accordingly the whole lesion was resected, its length being 21 cm.

HISTOLOGICAL REPORT.—The histological appearance of the nerve suggested that the proximal lesion (*b*) was inflicted by traction, and that the distal lesion (*a*) had been produced by crushing of the nerve and involvement with scar tissue. At the proximal end of the lesion, 10 cm. above the distal end of the neuroma, the nerve-bundles were separated by dense fibrous tissue, and there was also fibrous thickeneing of the perineuria. The arteries at this level showed intimal thickening. The 10 cm. of nerve between this level and the distal end of the neuroma formed an 'elongated' neuroma, for throughout this length of nerve there were signs of rupture of the Schwann tubes, with neuromatous outgrowth of Schwann cells and regenerating nerve-fibres into the connective tissue. Thus, the nerve-bundles had undergone rupture, but at different levels, so that there was no sharply defined neuroma as after complete division of a nerve. The damage was, however, most severe at the level of the macroscopical neuroma. Throughout the lesion there was dense fibrosis, endarteritis of the blood-vessels (*Fig.* 237), and lymphocytic infiltration of the tissue (*Fig.* 238): macrophages containing iron pigment were also scattered through the nerve.

Nerve-bundles containing some regenerating myelinated fibres were present in the thin strand of tissue below the neuroma, but at the level of the distal 'crush' lesion the nerve was completely obliterated and represented only by fibrous tissue.

THE PATHOLOGY OF THE NERVE LESIONS

In Platt's series of 9 cases 1 case was merely a transient block (neuropraxia) and complete recovery occurred in four weeks. The remaining 8 cases were subjected to operative exploration. In 3 cases the nerve-trunk was intact " with little or no naked-eye evidence of any lesion ". Of these three, one made a perfect spontaneous recovery, one made no recovery at all, and the third made a partial recovery—" sensory recovery complete; motor recovery partial ". In 5 cases there was complete rupture of the nerve-trunk and resection and suture was performed.

In our series, 4 cases (*Cases* 2, 4, 6, and 8) showed complete rupture of the nerve-trunk, though macroscopical continuity was maintained to a varying extent by a band of scar tissue between the stumps. In the remaining 4 cases there was continuity of the nerve-trunk: in *Case* 5 nerve-bundles were continuous throughout the lesion, and there was little or no evidence of rupture of the Schwann tubes at any level. In *Cases* 1 and 3 nervous tissue was continuous throughout the lesion, but in the latter and probably also in the former there had been some rupture of Schwann tubes at various levels. In *Case* 8 nervous tissue was continuous throughout the traction lesion, but probably all the Schwann tubes were ruptured, though at different levels, and the nerve was 'neuromatous' over a length of some centimetres. In *Case* 7 there was complete rupture of all the Schwann tubes at one level, but continuity of the nerve was maintained by an intact epineurium and a fusiform neuroma was produced. Thus, it appears that following severe traction there is considerable variation in the degree of disruption of the nerve which takes place. In about half of the cases in both Platt's and our own series the lesion amounted to complete division. In the remaining cases in our series macroscopical continuity was maintained and nervous tissue was present throughout the lesion, but the endoneural connective-tissue sheaths of the nerve-fibres, which remain as Schwann tubes after fibre degeneration, had in most cases undergone rupture at various levels. In one case there was no evidence of rupture even of the Schwann tubes.

Apart from the varying degree of injury to the connective-tissue sheaths we found certain constant pathological changes in the lesions:—

1. Extensive Degeneration of the Nerve-fibres.—In all 8 cases there was evidence of complete degeneration of axons and myelin throughout the resected length of nerve, and even in *Cases* 5 and 8, where the proximal resection was 20 cm. above the head of the fibula, the nerve-fibres had undergone degeneration up to the proximal limit of resection. Thus we cannot state how far up the nerve the degeneration takes place in these traction lesions, but it is certainly for a considerable distance proximal to the level at which the macroscopical appearance of the nerve is normal.

In all our cases in this series there was no sign of spontaneous recovery, though periods of from four to eleven months were allowed to elapse before operative interference. This failure is understandable in those cases in which the nerve was completely divided, as it was due to the inability of the regenerating fibres satisfactorily to bridge the gap between the stumps. But in the cases in which the nerve-trunk retained a considerable degree of continuity, the absence of recovery cannot be accounted for so simply. It cannot have been due to damage to the central neurons or to failure of the axons to regenerate through the lesion, for in all cases in which there was some continuity of the nerve very many non-myelinated axons were found to have grown out to its distal end. The most significant observation is that these axons had failed to increase to a normal diameter, and most of them had failed to become myelinated, and both these processes are essential conditions for a satisfactory recovery (*see* Young, 1942).

The reason for this failure of normal regeneration of the fibres, and thus for the failure of recovery, must probably be sought in the pathological changes described in the non-nervous tissues of the nerve-trunk at the site of the lesion.

2. Pathological Changes in the Blood-vessels and Connective Tissue.—The extensive

proliferation of collagen in the damaged nerves must have been the result of inflammatory changes consequent on the lesion: it cannot have been due to continued traction as the nerve cannot have been under tension for long after the injury. The pathological changes in the arteries and the signs of persistent œdema and lymphocytic infiltration suggest that the inflammatory processes are chronic, a condition which might account for the extensive 'interstitial neuritis', and for the failure of regeneration. If this interpretation is correct, then earlier operation on these lesions might have prevented the spread of fibrosis and nerve injury, and less extensive resections would have been necessary.

It may be mentioned that Stopford (1918) has described a case in which a sciatic-nerve lesion was followed by endarteritis of many of the principal vessels of the lower limb, and he attributed this pathological change to damage to the vasomotor fibres in the nerve at the level of the lesion.

FAILURE OF RECOVERY AFTER RESECTION AND SUTURE

In our series of lateral popliteal traction lesions suture was impossible in two cases (*Cases* 5 and 8), but possible in the remaining six. In *Case* 6 a two-stage suture was performed. In *Case* 7 the suture is too recent for any recovery to be expected. From *Table I* it will be seen that although in the remaining five cases a sufficient time has elapsed for recovery to take place, there has been evidence of recovery in only one of them (*Case* 4)

Table I.—Traction Injuries of the Lateral Popliteal Nerve: Recovery after Resection and Suture

Case	Delay before Suture	Site of Suture	Length Re-sected	Time of Assess-ment	Re-covery
1	6½ mth.	1·5 cm. above head of fibula	11·5 cm.	9 mth.	Nil
2	10 mth.	1·5 cm. below head of fibula	15 cm.	9 mth.	Nil
3	4 mth.	1 cm. above head of fibula	11 cm.	14 mth.	Nil
4	8 mth.	Neck of fibula	6 cm.	9 mth.	M.3, S.2
6	Bulb suture, 4 mth., suture, 10 mth.	6 cm. above head of fibula	Total, 14·8 cm.	9 mth.	Nil

In Platt's series resection and suture was performed in five cases. Two made a complete recovery, one made a partial recovery of motor and sensory functions, and two proved complete failures.

Progress in Case 4.—

Sept. 24, 1941: Resection and suture was performed. The extent of resection amounted to only 6 cm. (*see* p. 217).

May 13, 1942: Definite recovery of pain sensibility and sweating over the upper half of the lateral popliteal area.

June 16: Definite motor recovery in extensor longus digitorum (power 3), peroneus longus (power 1½), and tibialis anterior (power 1½). (The notation of voluntary power is that adopted by the Nerve Injuries Committee of the Medical Research Council.) There was also considerable recovery of pain sensibility and sweating.

Sept. 9: There was further recovery: tibialis anterior (power 3), extensor longus digitorum (power 3), peroneus longus (power 3), and peroneus brevis (power 2). There was almost complete recovery of pain sensibility and a marked recovery of touch sensibility and sweating.

In the cases which failed to show signs of recovery re-exploration was performed.

Re-exploration in Case 1.—

Oct. 16, 1941: The lateral popliteal nerve was palpable as a hard cord medial to the tendon of biceps. There was no palpable neuroma or tenderness of the nerve-trunk. The nerve could be pinched without causing tingling in the foot.

Jan. 23, 1942: Seven months after nerve suture, no evidence of motor or sensory recovery.

Second Operation (Jan. 23).—Exploration of the suture line. Local anæsthesia.

There was dense scarring of the whole operative field and freeing of the nerve-trunk was difficult and tedious.

State of the Nerve (*Fig.* 239).—Immediately below the sciatic bifurcation, 11 cm. above the head of the fibula, there was a slight thickening, the diameter of the

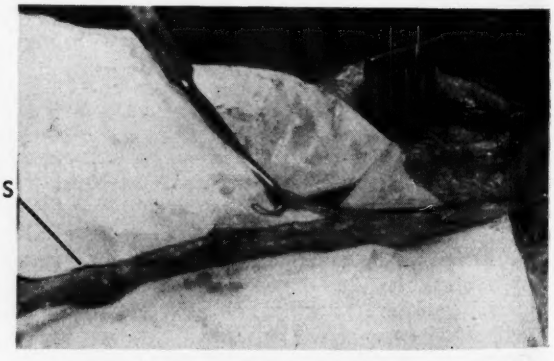

Fig. 239.—*Case* 1. The nerve exposed at the second operation, January 23, 1942. S, Suture line.

nerve being 9 mm. Below this the nerve was of constant diameter (6 mm.) as far as the original suture line. The suture line was identified by the tips of three black-silk sutures at 1·5 cm. above the head of the fibula. The nerve was somewhat thickened at this level, but there was no excessive induration at the suture line. The distal trunk measured 6 mm. in diameter; thus, the proximal and distal trunks were of equal diameter. The consistency of the whole nerve below the sciatic bifurcation was slightly firmer than that of the normal medial popliteal nerve. There was no macroscopical evidence of rupture at the suture line.

Stimulation: With bipolar faradic stimulation there was no motor response. Distal to the suture line a positive sensory response was obtained with a very strong stimulus. Above the suture line the

threshold at which a response was obtainable gradually decreased as the electrodes approached the sciatic bifurcation. Over the whole course of the nerve the threshold was greater than in the case of the normal medial popliteal nerve.

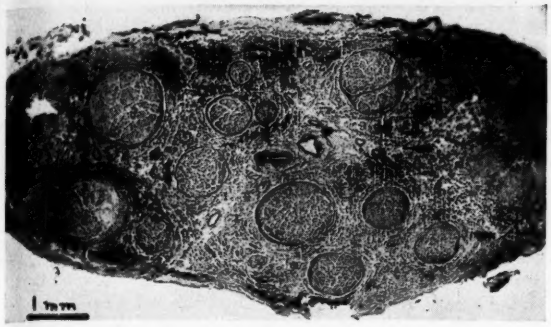

FIG. 240.—*Case* 1. The proximal end of the nerve resected at the third operation, March 11, 1942.

Procedure.—The appearance and consistency of the nerve suggested that there was no separation at the suture line, and the results of stimulation were taken to indicate that slight regeneration was in progress. However, biopsy was performed as follows: two small wedges of the nerve-trunk, about 1 cm. in length and 1·5 mm. in cross-section, were removed from 2 to 3 cm. above and 1 to 2 cm. below the suture line.

HISTOLOGICAL REPORT.—The biopsy specimens consisted of dense fibrous tissue of an appearance similar to tendon, with the collagen fibres orientated along the long axis of the nerve. They contained no nervous tissue.

March 11, 1942 (8½ months after nerve-suture): No evidence of motor or sensory recovery.

THIRD OPERATION (March 11).—Resection of the lateral popliteal nerve. Local anæsthesia.

Procedure.—On trial section of the nerve at the suture line, the trunk showed no macroscopical evidence of nervous structure. Accordingly, the lesion was resected to levels at which satisfactory nerve-bundles were visible. The proximal cross-section lay 14 cm. above the original suture line, the distal cross-section 5 cm. below the suture line. Total resection was thus 19 cm. The gap of 19 cm. was bridged with a nerve homograft removed, 30 minutes prior to its insertion, from a freshly amputated leg.

HISTOLOGICAL REPORT.—At the proximal end of the resected segment of nerve the fibrotic changes (*Fig.* 240) were similar to those seen in the proximal

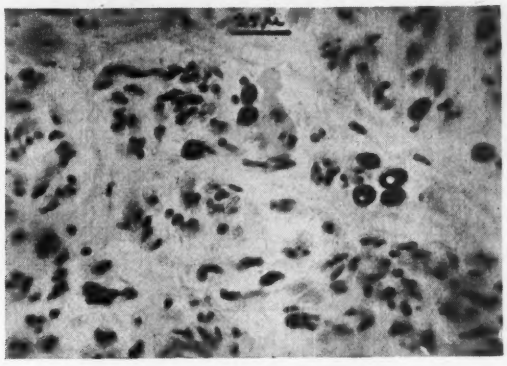

FIG. 241.—*Case* 1. Transverse section of part of a nerve-bundle from the proximal end of the nerve resected, March 11, 1942. Most of the regenerating fibres are of small diameter. (Weigert-carmine.)

stump at the suture line 8½ months earlier. The bundles were separated by epineural fibrosis, and some of them also showed collagen proliferation in the endoneurium. The nerve-bundles contained regenerating myelinated fibres, but these were as abnormal in number and diameter (*Fig.* 241) as at the suture point at the first operation (cf. *Fig.* 212). Thus regeneration had not progressed at all after the suture, and there had perhaps been retrograde degeneration. As the suture line was approached, the fibrosis in the nerve became increasingly severe: at a level about 10 cm. above the suture line some of the bundles were completely broken up and embedded in collagen (*Fig.* 242). The suture line could not be accurately identified in the sections, as fragments of suture material were found at various points over a length of 2 cm. or more in the nerve, and nervous tissue was continuous throughout the region. It thus appeared that a successful junction had been made between the stumps at suture, but the epineurium at least must have been considerably stretched as the remains of the sutures were so widely distributed.

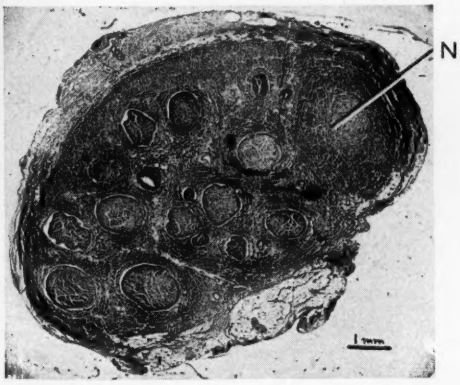

FIG. 242.—*Case* 1. Transverse section of the nerve resected, March 11, 1942, at a level 10 cm. proximal to the original suture line. One nerve-bundle (N) is completely broken up by fibrosis.

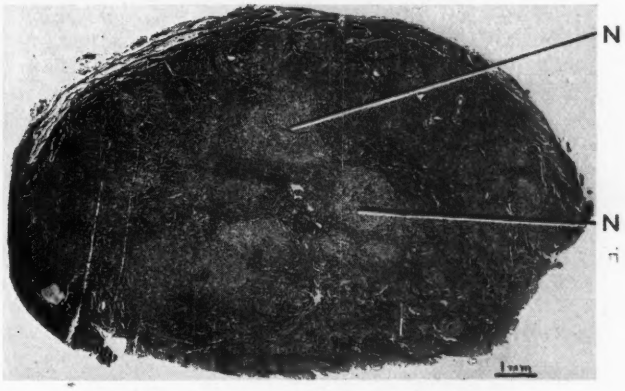

FIG. 243.—*Case* 1. Transverse section of the nerve resected, March 11, 1942, at the level of the original suture line. The nerve-bundles are completely broken up, but nervous tissue can be identified, as at N.

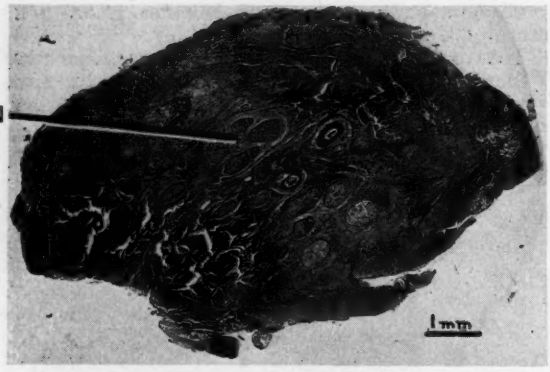

FIG. 244.—*Case* 1. Transverse section of the nerve resected, March 11, 1942, at a level distal to the suture line. Nerve-bundles (N) are visible, but much of the trunk is composed of dense fibrous tissue.

All around the level of the suture the nerve-bundles were completely broken up and the nerve consisted of a mass of collagen fibres, throughout which Schwann tubes were scattered singly or in small groups (*Fig.* 243). In the nerve distal to the suture some nerve-bundles were recognizable, but the fibrosis was more severe as the collagen was deposited more densely and was less cellular than in the proximal trunk (*Fig.* 244). The arterial trunks showed advanced intimal thickening, often with complete obliteration of the lumen of the vessel.

FIG. 246.—*Case* 2. Transverse section of part of a nerve-bundle from the proximal end of the nerve resected, March 25, 1942. The Schwann tubes contain only a few small myelinated fibres. (Weigert-carmine.)

Thus nerve suture had been followed by a reaction involving gross fibrous tissue proliferation, and there had been no further regeneration in spite of the fact that the suture line did not come apart during post-operative stretching.

Re-exploration in Case 2.—

Nov. 7, 1941: The nerve was palpable as a taut cord medial to the tendon of biceps (*Fig.* 215).

March 21, 1942: The nerve was no longer taut and could be moved from side to side with the knee fully extended. There was no evidence of motor or sensory recovery. Some shrinkage of the area of sensory loss was shown by procaine nerve-block to be due to overlap by neighbouring nerves, and not to true regeneration. Direct stimulation of the nerve was performed. An insulated unipolar needle electrode was inserted through the skin and applied to the nerve. Stimulation was negative at the level of the neck of the fibula, but positive 3 cm. above the head of the fibula (slight sensory response only).

SECOND OPERATION (March 25, 8½ months after nerve-suture).—Resection of the lateral popliteal nerve. Local anæsthesia.

As in *Case* 1, there was dense scarring around the nerve over the whole field of the previous operation.

State of the Nerve (*Fig.* 245).—The sciatic bifurcation lay 13·5 cm. above the head of the fibula. The appearance of the nerve was very similar to that of *Case* 1 at the exploration of Jan. 23, 1942. The site of suture was identified by three black dots of silk at the level of the head of the fibula. However, another black dot was identified 2 cm. above this, and indicated some elongation of the nerve at the suture line. The nerve felt normal at the sciatic bifurcation and for 5 cm. below it. It then became increasingly indurated, and at the level of the suture was somewhat flattened (diameter about 4 mm.). The distal trunk was also indurated, but the musculo-cutaneous and anterior tibial divisions, though atrophied, felt reasonably normal. There was no sign of neuroma formation.

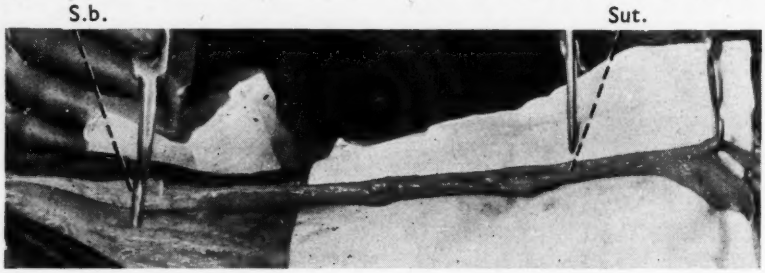

FIG. 245.—*Case* 2. The nerve exposed at the second operation, March 25, 1942. S.b., Sciatic bifurcation; Sut., Level of suture line.

FIG. 247.—*Case* 2. Transverse section of the nerve resected, March 25, 1942, at a level distal to the original suture line. A few nerve-bundles (N) are embedded in dense fibrous tissue.

Stimulation: Faradic stimulation—no motor response. No sensory response, except at the level of the sciatic bifurcation (but it it possible that the failure to elicit a response below this level was due to blocking of the nerve-trunk by the local infiltration).

Procedure.—Trial sections were made 1 cm. above and below the suture line. The sections showed no macroscopical evidence of nerve-bundles. Section 3 cm. above the suture line showed only a few tiny and poor nerve-bundles. Section 5 cm. above the suture line showed five insignificant gelatinous bundles and extensive perineural scarring. Section 7 cm. above the suture line was more satisfactory, but there was still extensive perineural scarring. Section 8 cm. above the suture line showed obvious nerve-bundles, but still with extensive scarring. Section 3 cm. below the suture line showed a few tiny bundles, but extensive scarring: it was noted that there was free bleeding from this cross-section. Section 4 cm. below the suture line showed only tiny bundles in both musculo-cutaneous and anterior tibial divisions, but further resection seemed futile. The total resection was 12 cm. in length: the gap was bridged by a homograft.

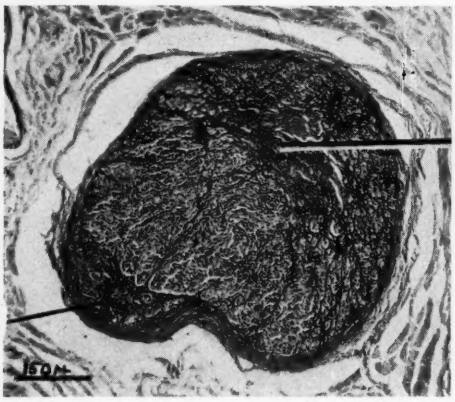

FIG. 248.—*Case* 3. Transverse section of the nerve resected, July 15, 1942, at a level 16 cm. proximal to the original suture line. Collagen has been formed within the bundle and on the inner side of the perineurium (C). (Masson.)

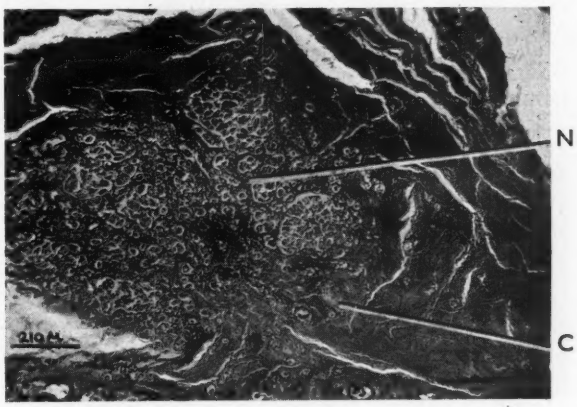

FIG. 249.—*Case* 3. Transverse section of the nerve resected, March 25, 1942, at a level approximately 8 cm. above the head of the fibula. The nervous tissue (N) is embedded in a dense mass of collagen (C). (Masson.)

HISTOLOGICAL REPORT.—At the proximal end of the resected nerve, 8 cm. above the original suture, the bundles were separated by epineural fibrosis. Its condition was similar to that of the proximal stump sutured at the first operation (*Fig.* 216), though the fibrosis was slightly more severe. The bundles contained some regenerating nerve-fibres, but they were fewer and smaller in diameter than they were at the suture line, 8 cm. below, 8½ months previously (*Fig.* 246, cf. *Fig.* 217). Thus, in the interval between suture and re-exploration there had been retrograde degeneration in the proximal stump of the nerve.

Over a distance of about 2·5 cm. at the level of the suture the nerve consisted entirely of a mass of collagen the fibres of which were chiefly orientated along the length of the nerve. In the trunk distal to this region some bundles were again seen, but they were embedded in dense collagen of which the trunk was largely composed (*Fig.* 247). The arteries showed considerable intimal thickening. In the proximal trunk the nerve was fibrosed throughout the length between the proximal level of resection and the remains of the suture line.

It seems likely that the sutured stumps did not separate immediately after operation, as the nerve felt taut during post-operative stretching, and macroscopical continuity was maintained. It is more likely that the stretching of the nerve caused gradual rupture of the Schwann tubes at the suture line some time after union had been effected. This gap gradually increased during stretching or while the limb was free, and it was progressively filled by collagen deposited by fibroblasts along the line of stress in the nerve.

Re-exploration in Case 3.—

March 10, 1942: No evidence of recovery. An insulated needle was passed through the skin and applied to the nerve. Unipolar faradic stimulation at the level of the head of the fibula proved entirely negative.

July 15: Still no evidence of motor or sensory recovery: the patient consented to re-exploration.

SECOND OPERATION (July 15, 15½ months after nerve suture).—Resection of the lateral popliteal nerve. General anæsthesia.

There was an enormous mass of scar tissue over the whole field of the previous exploration—even denser than in *Cases* 1 and 2.

State of the Nerve.—The nerve was identified at the level of the neck of the fibula, where both anterior tibial and musculo-cutaneous branches were greatly atrophied. The distal trunk was traced upwards to a level 2 cm. above the head of the fibula. Above this level there was no recognizable nervous tissue until about 10 cm. above the head of the fibula. In the 8-cm. interval the nerve seemed to consist entirely of scar tissue, but no trial sections were made.

Stimulation: Faradic stimulation proved entirely negative. The nerve was resected over a distance of 18 cm., from 2 cm. below the head of the fibula to 16 cm. above it.

The incision was closed and tenodesis of tibialis anterior and peroneus longus was carried out.

HISTOLOGICAL REPORT.—At the proximal end of the resected nerve, some 16 cm. above the original suture, the nerve-bundles were separated by epineural fibrosis, and blocks of collagen were deposited within them (*Fig.* 248). Some regenerating myelinated fibres were present in the nerve, but they were as few and as small in diameter as at the suture line 15½ months previously (*see Fig.* 220). Passing down the nerve towards the suture line the fibrosis in the endoneurium, perineurium, and epineurium of the nerve became more severe, till at a level 8 cm. above the

head of the fibula the nervous tissue was in the form of groups of Schwann tubes amongst blocks of collagen (*Fig.* 249). Through the next 6 cm. of the nerve, round the level of the suture, one or two small and abnormal nerve-bundles could be seen, but the trunk consisted chiefly of fibrous tissue. At the distal end of the resected segment more nerve-bundles were visible, but there was dense collagenization of all the connective-tissue sheaths. The arterial trunks showed advanced intimal proliferation, and there were some lymphoid aggregates around the blood-vessels.

Nerve suture had been followed by an extensive and massive deposition of collagen withing the nerve, with almost complete obliteration of nervous tissue for a considerable distance around the suture line.

FACTORS DETERMINING RECOVERY AFTER NERVE SUTURE

The absence of recovery after suture in *Cases* 1, 2, 3, and 6, and the pathological changes found in the sutured nerves at re-exploration of *Cases* 1, 2, and 3 belied the successful prognosis we had given from inspection of the condition of the sutured stumps. It thus seemed that we had neglected some important factor or factors governing the success of nerve-suture. The factors known to determine recovery after suture may be grouped as follows :—

1. The Condition of the Sutured Stumps.—The resection of the lesion should be sufficient to remove the proximal neuroma and distal glioma from the damaged nerve. A proximal trunk resected only to a level at which there is neuromatous criss-crossing of the fibres may provide outgrowing axons which will reach the distal stump, but there is a likelihood that many of them will not do so because of the devious pathways they follow in the proximal stump. In all the present series the resection had been adequate in this respect.

The resection should also be to a level at which the fibrosis between the nerve-bundles is not excessive. Excessive fibrosis in the distal stump is probably the more serious danger, as those fibres from the proximal stump which encounter scar tissue in the distal stump may cause the formation of a neuroma at the suture line. Interfascicular fibrosis in the proximal stump is perhaps less dangerous, as we have seen gunshot lesions in which the proximal stump contained many large myelinated fibres in spite of considerable fibrosis, and in such cases it seems probable that regeneration would not be hindered. In all our cases there was some interfascicular fibrosis, but it was not more severe than we have seen in stumps after gunshot lesions where a good recovery has followed suture. Moreover, in *Case* 4, in which the fibrosis was of the same grade as in the other cases, a good recovery is taking place.

The scar formed at the suture line must affect the growth of fibres into the distal stump. However, in the Oxford series of nerve-sutures an almost constant technique has been employed : the suture line was not under tension at the time of suture, only epineural sutures were inserted, and the suture material was very fine black silk except in a few cases where human hair or plasma was employed. Accordingly the scar formed at the suture line should be minimal in all cases.

For successful re-innervation of the distal stump and for recovery it is necessary that the proximal stump should provide an outgrowth of regenerating fibres. Fibre degeneration after a nerve lesion occurs over a variable distance proximal to the level at which there is macroscopical evidence of injury, and regeneration begins at the level where the retrograde degeneration ceases. Outgrowth into the distal stump can only commence when the fibres have reached the level of the suture line. We have shown that in these traction lesions the retrograde degeneration is very extensive, and even after

FIG. 250.—*Case* Mc.4 (*Table III*). Transverse section of a nerve-bundle at the proximal end of the resected lesion. Most of the myelinated fibres are of large diameter. (Weigert.)

extensive resection several months after the injury there were only few and small regenerating fibres in the sutured stump. This may influence recovery in two ways. First the paucity of fibres in the proximal stump may indicate that some unknown factor is impeding regeneration at a level well above the macroscopical lesion. Secondly, if regeneration to the level of the suture line is delayed, the fibres may find their pathway blocked by scar tissue when they reach it. The figures representing the number and size of the fibres in the stumps sutured after the resection of the traction lesions (*Figs.* 212, 217, 220, and 223) may be compared with *Fig.* 250, which shows the myelinated fibres in the proximal stump after resection of a lateral popliteal gunshot lesion (*Case* Mc.4, *Table III*). The fibres are much larger in diameter and more numerous than in the stumps of the traction lesions. However, the recovery in *Case* 4, where the fibres were equally sparse, suggests that this factor cannot be of paramount importance. Under suitable conditions the proximal stump in a traction lesion can provide an outgrowth of fibres adequate for recovery, even though it contains very few at the time of suture.

2. Delay before Suture.—It has often been stressed that long delay between injury and repair of peripheral nerves may prejudice the chances of successful recovery. Platt (1940) drew the

following conclusion from his series of sutures in traction injuries of the lateral popliteal nerve: "Although . . . the influence of the unknown factor—i.e., the degree of intraneural damage to axons in the proximal part of the nerve-trunk—cannot be discounted, these cases illustrate the well-established fact that early repair in a complete nerve lesion is of paramount importance in determining the quality of regeneration."

It is probable that in most peripheral nerve injuries the harmful effect of delay in repair results chiefly from progressive atrophy and fibrosis of the denervated end-organs. Experimental work has shown (Holmes and Young, 1942) that delay of more than a year does not affect the ability of the proximal stump to send out regenerating nerve-fibres at suture; and while changes in the distal stump affect the quality of recovery, they do not prevent it, even after a delay of two years.

There was a delay of several months before suture in our series of lateral popliteal traction lesions, but, as *Tables III–VIII* show, similar delays have not prevented recovery after other lesions. In the first 50 cases of nerve suture performed at the Oxford Centre the average delay was about seven months, yet in all but a few a satisfactory recovery is taking place. But it must be emphasized that such relatively short delays may be more harmful after traction lesions than in other injuries. For if the fibrotic and vascular pathological changes are progressive, then early operation on these lesions might be much more successful.

3. Level of Suture.—Stopford (1920) claimed that sutures in the distal part of a limb are less successful than those in the proximal part. From the cases listed in the tables it does not seem that this factor is sufficiently weighty to account for the complete failure of four of our lateral popliteal sutures.

4. The Extent of Resection before Suture.—We consider that the failure of recovery in our *Cases* 1, 2, 3, and 6 was primarily due to the lengthy resection necessary before suture and the consequent extensive post-operative stretching of the nerve. The failure cannot have been simply due to rupture of the suture line by too short immobilization or too hasty stretching. The whole limb was immobilized for three weeks after suture, and experimental work (Miller, 1921) has shown that the strength of a suture line in nerve—its ability to resist rupture—increases to a maximum in the first three weeks after suture. And Highet and Sanders (1943) have shown that an experimental procedure similar to that adopted in our cases does not cause rupture of the suture line, though there is intraneural damage. We have subjected to re-exploration two cases of nerve suture which failed to show recovery because of complete disruption at the suture line. The macroscopical and histological appearance of these nerves was quite unlike that in our three cases described above. *Fig.* 251 shows an obvious disruption of the suture line in a case of suture of the lateral popliteal nerve performed at another hospital, where the limb was immobilized for only two weeks after suture and then allowed free movement.

From comparison of *Cases* 1, 2, and 3, with *Case* 4 we conclude that resection of more than about 9 cm. of the lateral popliteal nerve is not

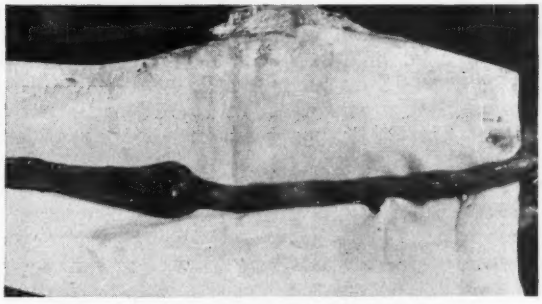

FIG. 251.—The nerve exposed at operation in a case in which the suture line had been ruptured as a result of too rapid stretching (*see* text). There is a proximal neuroma connected to the distal nerve by a band of scar tissue.

compatible with recovery. The post-operative stretching of the nerve, no matter how carefully it is carried out, inflicts irreparable damage on the nerve in the form of retrograde degeneration of the fibres, arterial and inflammatory changes, and massive intraneural fibrosis.

THE EXTENT OF RESECTION COMPATIBLE WITH RECOVERY AFTER END-TO-END SUTURE

Forrester-Brown (1921) and Babcock (1927), in describing the surgical technique for the closure of large gaps in peripheral nerves by end-to-end suture, have listed the maximum resections possible in various nerves (*Table II*). Unfortunately Babcock does not give any account of the recovery which followed these large resections, and Forrester-Brown's figures are based on a much smaller number of cases, and their recoveries were not all followed up. The possibility that post-operative stretching may cause damage to the nerve is stressed by Leriche (1940) and Stookey (1922). Stookey refers to 2 cases of bulb suture in which post-operative stretching was carried out as a preliminary to a second-stage

Table II.—THE MAXIMUM LENGTH OF RESECTION OF VARIOUS PERIPHERAL NERVES COMPATIBLE WITH END-TO-END SUTURE
(From *Forrester-Brown*, 1920, *and Babcock*, 1927)

NERVE	FORRESTER-BROWN	BABCOCK
Median in arm	11·5 cm.	15 cm.
Median in forearm	16·5 cm.	23 cm.
Ulnar in arm	12·5 cm.	16 cm.
Ulnar in forearm	16·5 cm.	12·5 cm.
Radial in arm	9–10 cm.	15 cm.
Sciatic	10 cm.	14 cm.
Posterior tibial below mid-leg	15 cm.	13–15 cm.

end-to-end suture. In both cases "intraneural hæmorrhages had occurred, probably throughout a great extent of the nerve-trunk, with subsequent scar formation and sclerosis". He expressed the opinion that "nerve stretching may be permitted only up to the point of taking up the normal laxity of the nerve-trunk. If greater stretching be applied, there is a danger of grave injury to the nerve, proportionately greater, the greater the degree of stretching."

We have analysed all cases of nerve-suture performed at the Oxford Peripheral Nerve Injury Centre in an attempt to determine whether there is any correlation between the extent of the resection and the degree of recovery. We have excluded all recent cases where the period of observation is too short for any recovery to have taken place. We have also excluded the few cases in our series in which no adequate measurement of the amount of resection was noted. All the cases are too recent for a final assessment of recovery, but the recovery up to the present date is recorded.

The assessment of recovery in peripheral-nerve injuries is rather difficult. The statistics of recovery in nerve injuries treated in the last war were so compiled that it is practically impossible to compare the results published by any two clinics. Recovery may be assessed either from a neurological or economic standpoint. In the Jacksonian Essay, 1941, one of us (W. B. H.) suggested a scheme of assessment on a neurological basis. This scheme has been adopted with minor modifications by the Nerve Injury Committee of the Medical Research Council. In brief the scheme is as follows :—

1. *Motor Recovery* :—

Stage 0.—No contraction.

Stage 1.—Return of perceptible contraction in the proximal muscles.

Stage 3.—Return of function in both proximal and distal muscles of such an extent that all important muscles are of sufficient power to act against resistance.

Stage 4.—Return of function as in Stage 3, with the addition that all synergic and dissociated (isolated) movements are possible.

Stage 5.—Complete recovery.

2. *Sensory Recovery* :—

Stage 0.—Absence of sensibility in the autonomous zone.

Stage 1.—Recovery of deep cutaneous pain sensibility within the autonomous zone.

Stage 2.—Return of some degree of superficial cutaneous pain and touch sensibility within the autonomous zone

Stage 3.—Return of superficial cutaneous pain and touch sensibility throughout the autonomous zone, with disappearance of any over-response.

Stage 4.—Return of sensibility as in Stage 3, with the addition that there is recovery of two-point tactile discrimination within the autonomous zone.

In *Tables III–VIII* 42 cases of nerve suture are analysed. As this is only an interim assessment we have not examined all the factors concerned in recovery, such as the nature of the injury and the histological state of the sutured stumps.

The tables show that in the majority of peripheral-nerve lesions adequate resection may be performed with the expectation of recovery after suture. All of the cases listed in the tables followed gunshot or lacerated or incised wounds, and none of them followed a traction injury. Of the 42 cases, some recovery has been noted in all but 3 (2 lateral popliteal sutures P.3 and R.3., and 1 median suture M.5). In both the lateral popliteal cases suture had been performed previously at another hospital and our suture was performed at a re-exploration carried out when recovery failed to take place. Consequently, in *Case* R.3, 16 months had elapsed after the

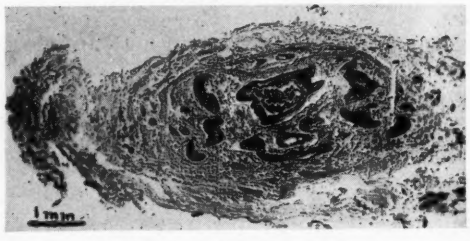

FIG. 252.—*Case* R.3 (*Table III*). Transverse section of the distal end of the resected lesion. The bundles are of abnormal appearance and embedded in fibrous tissue.

Table III.—LATERAL POPLITEAL SUTURES
(6 *cases*)

CASE	DELAY BEFORE SUTURE	SITE OF SUTURE	LENGTH RESECTED	TIME OF ASSESSMENT	RECOVERY
W.14	6 mth.	Head of fibula	9 cm.	7 mth.	M.1. S.0.
M.40	Resection of nerve tumour (Schwannoma). Immediate suture	5 cm. above head of fibula	9 cm.	7 mth.	M.1. S.0.
Mc.4	6½ mth.	Head of fibula	7·8 cm.	9 mth.	M.1. S.1.
P.3	9 mth.	15 cm. above head of fibula	7 cm.	17 mth.	Nil
R.3	16 mth.	Neck of fibula	5·5 cm.	14 mth.	M.0. S.1.
B.9	4 mth.	9 cm. above head of fibula	5 cm.	16 mth.	M.3. S.2.

original injury when our suture was performed, and after our resection and suture the histological examination of the resected lesion showed that the peripheral stump sutured was of a very abnormal appearance (*Fig.* 252), and resection had apparently been inadequate. In *Case* M.5 the resection was 9 cm.—greater than in the other cases of median suture which proceeded to recovery.

In our series it will be seen that the maximum resections performed are: Combined sciatic, 8·5 cm; medial popliteal, 6 cm.; median, 9 cm.; ulnar, 11 cm.; radial, 6 cm. In the lateral popliteal traction series the maximum resection was 15 cm. Thus it is only in the repair of the lateral popliteal traction injuries that the extent of our resections have approached the maxima proposed by Babcock. In the median case, M.5,

Table IV.—Sciatic Sutures (Combined Medial and Lateral Popliteal) (3 *cases*)

Case	Delay before Suture	Site of Suture	Length Resected	Time of Assessment	Recovery
B.3	6 mth.	25 cm. above knee	8·5 cm.	17 mth.	(Both components) M.1. S.1.
L.1	3 mth.	22 cm. above knee	7 cm.	15 mth.	(Both components) M.1. S.1.
M.4	8 mth.	20 cm. above knee	6 cm.	17 mth.	(Both components) M.1. S.1.

Table V.—Medial Popliteal Sutures (2 *cases*)

Case	Delay before Suture	Site of Suture	Length Resected	Time of Assessment	Recovery
C.22	11 mth.	14 cm. above head of fibula	6 cm.	15 mth.	M.1. S.0.
B.11	10 mth.	9 cm. below head of fibula	6 cm.	13 mth.	M.1. S.1.

Table VI.—Ulnar Sutures (12 *cases*)

Case	Delay before Suture	Site of Suture	Length Resected	Time of Assessment	Recovery
S.4	11 mth.	13 cm. above medial epicondyle	11 cm.	11 mth.	M.2. S.2.
C.21	10 mth.	13 cm. below medial epicondyle	9·5 cm.	12 mth.	M.1. S.1.
C.30	4½ mth.	15 cm. above medial epicondyle	9 cm.	9 mth.	M.1. S.0.
M.20	10 mth.	10 cm. above medial epicondyle	8 cm.	10 mth.	M.2. S.2.
H.8	10 mth.	7 cm. below medial epicondyle	7 cm.	12 mth.	M.1. S.1.
D.8	8½ mth.	9 cm. above medial epicondyle	5 cm.	8 mth.	M.1. S.0.
F.4	7½ mth.	8·5 cm. above medial epicondyle	4 cm.	17 mth.	M.2. S.2.
M.17	2½ mth.	8 cm. below medial epicondyle	4 cm.	16 mth.	M.3. S.2.
C.20	4 mth.	At wrist	4 cm.	13 mth.	M.2. S.1.
M.26	5 mth.	At wrist	4 cm.	8 mth.	M.3. S.2.
T.3	8 mth.	At wrist	3 cm.	15 mth.	M.3. S.2.
A.5	1½ mth.	At pisiform	1·5 cm.	18 mth.	M.3. S.3.

the resection was 9 cm.; Babcock's maximum for resections of the median at this level is 15 cm., but in case M.5 no recovery has taken place.

Thus we conclude that with the exception of the median and lateral popliteal nerves our resections have not been sufficiently extensive to be incompatible with recovery. Yet the fact that no recovery has followed a resection of 11 cm. and more in the lateral popliteal, and 9 cm. in the median, suggests that resections of the extent that Babcock considers possible are incompatible with recovery. It cannot be emphasized too much that the recording of details such as the extent of resection and the site of suture at every operation on a peripheral nerve lesion is essential for a proper correlation of operative procedure with functional recovery. It is probable that the maximum resection compatible with recovery depends not only on the nerve, but on the site of suture. If the suture line overlies an acutely flexed joint the degree of damage during post-operative stretching may be more severe.

The results of treatment in our series of cases have been most unsatisfactory. Only in *Case* 4 is a satisfactory recovery taking place. *Case* 7 may show some recovery, but it is unlikely to be complete as the condition of the sutured stumps seemed unsatisfactory. Treatment of lateral popliteal traction lesions thus presents a difficult problem. When, as in *Case* 4, the extent of the resection necessary is not too great a good recovery may be expected, but when a

Table VII.—MEDIAN SUTURES
(12 *cases*)

CASE	DELAY BEFORE SUTURE	SITE OF SUTURE	LENGTH RESECTED	TIME OF ASSESSMENT	RECOVERY
M.5	11 mth.	6 cm. above medial epicondyle	9 cm.	15 mth.	Nil
C.30	4½ mth.	16 cm. above medial epicondyle	7 cm.	13 mth.	M.1. S.0.
F.6	10½ mth.	6·5 cm. below coracoid process	6·2 cm.	12 mth.	M.1. S.1.
M.3	8 mth.	11 cm. above wrist	7 cm.	10 mth.	M.2. S.2.
C.17	10 mth.	6·5 cm. above wrist	7 cm.	14 mth.	M.2. S.2.
S.15	7 mth.	At wrist	5 cm.	6 mth.	M.2. S.1.
M.26	5 mth.	At wrist	5 cm.	8 mth.	M.3. S.3.
P.17	3½ mth.	4 cm. above wrist	3·2 cm.	8 mth.	M.2. S.2.
C.1	4½ mth.	At wrist	3 cm.	12 mth.	M.2. S.3.
T.3	8 mth.	At wrist	3 cm.	15 mth.	M.3. S.2.
D.12	2½ mth.	At wrist	2·5 cm.	16 mth.	M.3. S.3.
M.21	1 mth.	At wrist	2 cm.	5 mth.	M.0. S.2.

Table VIII.—RADIAL NERVE SUTURE
(7 *cases*)

CASE	DELAY BEFORE SUTURE	SITE OF SUTURE	LENGTH RESECTED	TIME OF ASSESSMENT	RECOVERY
S.9	12 mth.	14 cm. above lateral epicondyle	6 cm.	12 mth.	M.1. S.1.
M.8	Bulb suture, 7½ mth.; 2nd stage, 10 mth.	9 cm. above lateral epicondyle	6 cm.	11 mth.	M.2. S.1.
B.4	6 mth.	5 cm. above lateral epicondyle	5·5 cm.	14 mth.	M.3. S.3.
N.5	4 mth.	Lateral epicondyle	5 cm.	6 mth.	M.3. S.1.
F.5	4½ mth.	15 cm. above lateral epicondyle	4·7 cm.	14 mth.	M.2. S.1.
R.7	2½ mth.	Lateral epicondyle	4 cm.	12 mth.	M.3. S.2.
F.2	11½ mth.	17 cm. above lateral epicondyle	4 cm.	12 mth.	M.1. S.0.

more extensive resection is required the post-operative stretching inflicts irreparable damage on the nerve. The process of two-stage suture, as carried out in *Case* 6, does not seem to give any better results. And unless *Case* 7 shows recovery it seems unlikely that a deliberately incomplete resection of the lesion can be recommended.

Platt recommended early exploration, and it is true that in our series no recovery followed conservative treatment even in the cases in which the nerve had not undergone complete disruption. If it is true that the pathological changes in the nerve at the site of the traction lesion are progressive, as our pathological investigations suggest, then resection and suture within a few weeks of the injury might be possible without extensive resection. But in cases where the nerve is not completely disrupted it would be difficult to decide at an early exploration whether or not resection and suture was necessary. In Platt's 3 cases in which the nerve was explored early, macroscopical continuity was found, and resection was not performed, recovery was complete in one case, incomplete in the second, and absent in the third.

Our series is, unfortunately, too small to allow us to determine the best procedure, but taking Platt's cases also into account, it would appear that early exploration is indicated in all cases. If the nerve lesion amounts to complete division, resection and suture should be attempted. If macroscopical continuity is preserved and the nerve seems reasonably normal, nothing further should be done, but if the nerve is abnormal in appearance and to palpation, in spite of the macroscopical continuity, trial sections should be made at the centre of the lesion, and if extensive scarring is found, resection and suture should be performed. In cases where at either early or late exploration the resection necessary is extensive—10 cm. or more—suture is not justifiable and alternative measures such as nerve-grafting should be considered. Whatever view is taken of the value of nerve-grafts, the recovery cannot be worse than after resection and suture in such cases.

It is fortunate that the disability which results from a permanent complete lateral popliteal paralysis is less than that from any other major nerve injury, and that most patients return to useful work while wearing a satisfactory toe-raising spring.

SUMMARY

1. Eight cases of traction injury to the lateral popliteal nerve have been treated surgically and the lesions examined histologically.

2. The traction lesion is characterized by much more extensive damage to the nerve-trunk than is found after injury by incision, laceration, or gunshot wound. The pathological changes in the nerve extend far above the site of obvious damage, in the form of fibre degeneration, intraneural fibrosis, and vascular abnormality.

3. The connective sheaths of the nerve undergo rupture to a varying degree; thus the nerve may be completely divided, or bear a fusiform neuroma, or show macroscopical continuity.

4. Spontaneous recovery did not take place even in cases in which nervous tissue was continuous throughout the lesion. Regenerating nerve-fibres succeeded in crossing the lesion, but they remained small in diameter and most of them failed to become myelinated.

5. In three cases end-to-end suture was performed after an extensive resection of the lesion: closure of the gap was obtained by extensive mobilization of the nerve and flexion of the knee. No recovery took place in these cases. In one case in which a less extensive resection was necessary a good recovery is in progress.

6. A further exploration of the nerves which failed to show recovery after suture was carried out. Histological examination showed pathological changes similar to those following the original lesion, but even more severe and extensive.

7. It is concluded that failure of recovery after extensive resection and suture may be due to injury inflicted on the nerve during post-operative extension of the joint.

8. This and other factors affecting recovery after suture are discussed with reference to a short series of cases of peripheral nerve injuries of other types.

We owe our thanks to Professor H. J. Seddon and Mr. J. Z. Young for help in various ways. One of us (W. H.) holds a Beit Memorial Research Fellowship.

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THE TREATMENT OF PANCREATIC CYSTS

By JUDSON T. CHESTERMAN, Sheffield

The accepted method of treatment of large pancreatic cysts, not amenable to excision, has been marsupialization, which was first described by Gussenbauer in 1883. This is an unsatisfactory operation, as the fistula may discharge for years and frequently terminates in death from retroperitoneal sepsis and thrombophlebitis of the inferior vena cava. Jurasz (1931) was the first to anastomose the cyst to the stomach, and since then Harries (1934) has also reported success with this method.

Retroperitoneal anastomosis to the duodenum has been carried out by Kerschner (1935), but the following case is, as far as I know, the first record of drainage into the jejunum.

CASE REPORT

A. C., aged 39, housewife, Case No. 142638.

This patient first came under my care on Nov. 2, 1938, suffering from acute small-gut obstruction of eighteen hours' duration. Six weeks before she had had a vaginal hysterectomy for a prolapsed fibroid uterus. Laparotomy showed a right-sided ovarian cyst, the size of a tennis ball, with a loop of jejunum adherent to its pedicle. The loop was freed and the cyst was removed. No other abnormality, save some pelvic adhesions, was found.

She remained well until June 3, 1941, when she was taken with severe abdominal pain and vomiting, which became progressively worse, and she was readmitted on June 5. Immediate laparotomy, under spinal anæsthesia, by the resident surgical officer, showed blood-stained fluid, fat necrosis, and a large cyst in the region of the head of the pancreas. I was then asked to see her and closed the abdomen without drainage. On account of shock three pints of plasma and two pints of blood were given. Twenty-four hours later her condition was satisfactory, though slight obstructive jaundice developed, which lasted a week.

The cyst steadily increased in size, and by the end of June, in spite of her obesity, was readily visible from the bottom of the bed. Also at that time the leucocyte-count became normal, the glucose tolerance test showed a normal blood-sugar curve, with no glycosuria, and there was no evidence of deficiency in the external pancreatic secretion.

The X-ray report (June 30) of a barium meal was: Œsophagus, no appreciable disease. Stomach, normal size and position. The greater curve of the prepyloric segment is indented by the large rounded palpable mass which appears to be pancreatic in origin. The barium left the stomach slowly. The duodenal loop shows some enlargement, but by palpation the mass seemed to extend beyond it. At four hours the stomach was empty and the barium was in the colon and ileum, the former being depressed by the mass (*Fig.* 253).

Operation (July 9).—Anæsthetic, open ether. Upper right paramedian incision. The lesser sac was opened through the great omentum and a thick-walled cyst was aspirated of 42 oz. of slightly blood-stained fluid. The jejunum, about 2 ft. from the duodenojejunal flexure, was anastomosed to the cyst, a 2-in. stoma being made. The wound was closed in layers.

Progress.—Convalescence was uneventful and the patient was discharged on Aug. 2., symptomless.

The X-ray report (July 31) of a barium meal was: The compression of the stomach by the cyst is now considerably less, though still present to a slight degree. The meal passed readily through the small intestine, and none of it went through the anastomosis from the jejunum to the cyst. (*Fig.* 254.)

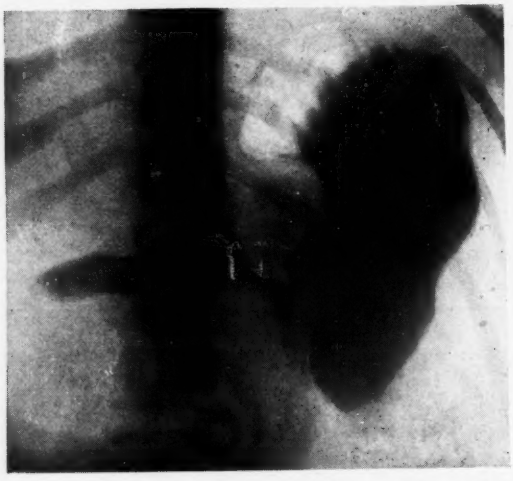

Fig. 253.—Pre-operative radiograph showing the marked displacement of the greater curve by the cyst.

Fig. 254.—Radiograph taken one month after operation, showing still some displacement of the greater curve by the cyst.

July 24, 1942. The patient has been in good health since the operation.

The X-ray report (July 20, 1942) of a barium meal was: Radiograph shows no pressure deformity of the greater curvature of the pre-pyloric region now. The size of the duodenal loop is unchanged. There is no apparent delay in the passage of barium, and barium could not be made to pass through the anastomosis of the jejunum to the cyst.

SUMMARY

A new method of treatment of pancreatic cyst is described. Anastomosis to the jejunum is recommended, as it is easier to perform than any other satisfactory method so far described, and it also approximates more to the principle of dependent drainage.

I wish to thank Dr. J. Rennie, Medical Officer of Health for Sheffield, and Dr. J. Clark, Medical Superintendent, City General Hospital, Sheffield, for allowing me to publish this case; and also Dr. E. K. Abbott for the X-ray studies.

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SPINAL ANALGESIA FOR PROSTATECTOMY

A REVIEW OF 94 CASES WITH NOVOCAIN DISSOLVED IN THE SPINAL FLUID

By R. P. HARBORD

DEMONSTRATOR IN ANÆSTHETICS, UNIVERSITY OF LIVERPOOL
VISITING ANÆSTHETIST, SMITHDOWN ROAD CITY HOSPITAL

To proclaim that spinal analgesia should be reserved for the robust healthy patient is as fallacious as to say that it is the ideal method for the poor risk. In my opinion it is especially indicated for the operation of suprapubic prostatectomy, but many object to its use on account of the danger of reactionary hæmorrhage.

Those with prostatic disease are usually emphysematous, suffer from chronic bronchitis, and have some degree of myocardial weakness. There is often a diminished renal function, and at operation brisk hæmorrhage may be encountered. General anæsthetics tend to enhance the latter during enucleation, and they may be accompanied by a period of excitement during the recovery stage when the patient attempts to remove his catheter.

SUMMARY OF RESULTS

During the past four years spinal anæsthetics were given to 94 suprapubic prostatectomies at Smithdown Road Hospital, Liverpool, 78 of which were personally conducted. Cases of prostatic obstruction treated by peri-urethral resection during the same period are not included, as in these a 'low' spinal anæsthetic was administered, involving a different technique.

The average age was 64 years, and 77 per cent were admitted with acute retention of urine. Prior to operation in 40 per cent the urea concentration test was unsatisfactory and in 47 per cent the non-protein nitrogen of the blood was 45 mg. per 100 c.c. or over.

There were no deaths on the table, although 2 died on the day of operation. One of these had a sudden epileptiform seizure three hours afterwards and was probably a cerebral hæmorrhage; the other was due to shock, but unfortunately I was not able to observe this case. The remaining 12 died at intervals of from 2 to 26 days. Six of these died of uræmia, 2 from heart failure due to myocarditis, and 1 developed bronchopneumonia. The total death-rate was therefore 15 per cent.

In 5 per cent there was considerable hæmorrhage during operation, and in 17 per cent troublesome bleeding was encountered on the next day, and in 1 case it went on for two days. None of the deaths was due to loss of blood, although it may have been a factor in 2 cases.

In 6 per cent the analgesia was unsatisfactory, necessitating supplementary anæsthesia by cyclopropane or gas-oxygen and minimal ether. There was one headache severe enough to cause a request for treatment. No instances of meningitis or post-spinal paralysis were recorded.

PRELIMINARY CONSIDERATIONS

No premedication was given except in very nervous subjects. The patients were nursed in a ward set apart for urinary cases. They were brought into the best possible condition before operation by rest and medical treatment, and in those with retention of urine the bladder was slowly decompressed. In 43 per cent a first-stage cystotomy was performed, mainly with local analgesia, as a preliminary to a Freyer operation. They had the experience of seeing other patients with similar conditions, with the result that they knew what to expect, and approached the operation without any unnecessary fear. The lack of premedication enabled more morphine to be given post-operatively, so that restlessness was not common.

In 56 per cent a modified Harris operation was performed, in which retrigonization of the prostatic cavity was carried out but the bladder was not closed, drainage being effected by a urethral catheter and a suprapubic tube. Suction drainage was provided in all cases.

The principles involved in the selection of cases for different methods of treatment are described elsewhere (Cosbie Ross, *Brit. Med. Jour.*, 1936, Dec.).

PRINCIPLES OF THE SPINAL TECHNIQUE

The drug used was novocain in powder form, and from 90 mg. to 150 mg. (usually 100 mg.) were dissolved in the spinal fluid. The dose of novocain varied according to the clinical risk and not with the size of patient.

Analgesia comes on rapidly, and this hyperbaric solution gravitates towards the fifth dorsal segment when the patient lies horizontally. The spread of analgesia is followed by testing sensation repeatedly until the umbilicus is reached (D.10), when the table is tilted into 5° of anti-Trendelenburg position, and the solution passes back over its original path. This is known as ' re-blocking ' and was first described by Vehers.

DETAILS OF THE TECHNIQUE

The patient lies on his side with the spine arched and the table horizontal. The anæsthetist cleanses the hands and dons sterile gown and gloves. A spirit swab is then applied to the back with a pair of sterile forceps. The skin is infiltrated in the middle line between a convenient pair of vertebræ well below the first lumbar vertebra. Five c.c. of ½ per cent novocain, to which has been added ½ gr. of ephedrine, usually allows a painless lumbar puncture with a Howard Jones needle. The skin is not touched with the gloved hands. This non-touch technique is not always possible when the surface markings cannot be seen, but it is attempted in the first instance.

A syringe then aspirates 5 c.c. of cerebrospinal fluid, and this is injected into an ampoule containing the required dose of novocain. The ampoule is not regarded as strictly sterile, although it has been immersed in spirit, and it is handled with a sterile gauze swab. The resulting solution is re-injected into the subarachnoid space slowly after first slightly withdrawing the plunger to ensure continuity. Care is exercised so that little or no fluid is wasted

Immediately following the injection the patient is turned on to his back and the towels removed from the abdomen. The method for testing skin sensation is important, as upon its appreciation depends the accuracy of the technique. After being told that he is to be tested, an intravenous needle is held so that it lightly pricks the skin near the pubic spine on one side of the middle line. Gradually the pressure on the needle is kept up until the operator is sure that pain would be felt in a normal skin. Another area is then chosen slightly higher up the abdomen. When light pricking results in no pain response, the needle is driven into the skin up to the end of the bevel. A slight muscular reflex twitch suggests the presence of an unanæsthetized area in a patient who will not co-operate. In this way the upward path of analgesia is followed.

When the umbilicus is reached the table is placed in the anti-Trendelenburg position and left in about 5° for two minutes.

In the normal spine in the supine position, and with the table horizontal, a hyperbaric solution will ascend in the subarachnoid space until it reaches the 5th or 6th dorsal segment of the cord. In the aged the curve of the dorsal vertebræ is less pronounced in many cases, and this is readily appreciated on doing the lumbar puncture. In these the table is put in about 5° of anti-Trendelenburg position immediately the spinal injection is completed.

By the process of re-blocking, the analgesic solution soaks the nerve-roots twice and tends to prevent a patchy distribution of the area. When the surgeon is ready, shoulder rests are securely fixed and about 10° of Trendelenburg tilt is given to the table. This results in the area being soaked thrice. By this time, when the solution reaches the tenth dorsal area most has been fixed by the tissues or absorbed into the blood. This is probably the correct interpretation, because the analgesia does not ascend any higher. In the early days when this method was being evolved, the patients were put head up, but with the table in about 10° Trendelenburg, and there were no cases of cervical involvement.

Since the distance from L.3 or 4 to the tenth dorsal segment is not more than a few centimetres, speed is essential in performing the tests for sensation. There should be the least possible delay in removing the towels from the abdomen.

At one time it was thought that the sensory analgesic area could be reliably estimated by testing the function of various muscles. For instance, if the patient was unable to flex his hips (iliopsoas, L.1, 2, and 3) it was assumed that the sensory area would be about two segments higher (i.e., D.10). This method was abandoned for two reasons : (1) When it was used for hernial operations in many cases satisfactory analgesia was present, although the patient was capable of flexing his hips ; (2) Sensation is a quicker test and therefore more reliable, for during the time lost the drug is ascending.

The modern tendency has been to employ hypobaric solutions in preference to the hyperbaric ones. The disadvantages of light percaine is that the analgesia takes much longer to come on, sometimes not being present for ten or more minutes after the injection. Precision by sensation methods is therefore out of the question. Spinocain analgesia is quicker, but does not appear so certain in its effects. Also I have occasionally observed the height of analgesia to rise in patients in the Trendelenburg position, indicating that the novocain has dissociated from the alcohol fraction of the spinocain. Heavy percaine (1–200) is not so rapid in action as novocain and the latter is therefore preferable.

COMPLICATIONS AND THEIR TREATMENT

The fall in blood-pressure is not usually marked when the upper margin of analgesia has been at the umbilicus. Those with a high pre-operative blood-pressure usually exhibited a fall with or without nausea. Whenever a severe fall in blood-pressure was detected, whether there were symptoms or not, an intramuscular injection of 1 c.c. of veritol was given and oxygen inhalation with a B. L. B. mask commenced. Coramine, pituitrin, and ephedrine are not reliable in treating this condition. This was all that was required, except where there had been much blood-loss. In these cases a blood transfusion is indicated.

SUMMARY AND CONCLUSIONS

A series of 94 cases of the poor-risk type have been given spinal analgesia, with novocain dissolved in cerebrospinal fluid, for prostatectomy. There were 14 deaths, 2 on the same day and 12 at later dates.

A technique is described in which a sensation test is used to follow the upward path of analgesia, with control by the process of re-blocking when a sufficient area is affected.

In most cases it is possible to obtain an analgesic area up to the umbilicus and no higher by using the technique described.

Associated with this level there may be a fall in blood-pressure due mainly to vasomotor paralysis, and this may be adequately treated by veritol injection and oxygen inhalation.

Spinal analgesia administered in a haphazard manner and without control is a lethal weapon, even in the normal patient, but given with an understanding of the principles of precision, is of great value in selected groups of poor risks.

I wish to thank Dr. Minnitt for much valuable advice; and Mr. J. Cosbie Ross, Visiting Surgeon and Urologist to Smithdown Road City Hospital, for constant encouragement during my attempts to perfect the method. I have also to thank Dr. Steel, Medical Superintendent, for permission to publish this work. Thanks are also due to Messrs. Evans, Lescher & Webb who supplied me with varying doses of novocain in ampoules sufficiently large to accommodate 5 c.c. of spinal fluid.

A CASE OF TRAUMATIC VENTRICULAR PNEUMOCEPHALUS

By D. OWEN DAVIES, Manchester

The presence of air within the cranial cavity following fracture of the skull has been the subject of numerous articles since it was first demonstrated radiologically by Luckett[1] in 1913. The first considerable review of the condition was by Dandy[2] in 1926, who collected 25 cases and added 3 of his own, while in 1932 Worms, Didiée, and Grumbach[3] produced a comprehensive analysis of 72 published cases with an extensive bibliography. The literature in this country is, however, scanty, and I have been able to trace little more than a dozen cases.[4–11]

The case here described presents a problem in the treatment of the condition when it is recognized immediately after the injury.

CASE REPORT

A man of 59 years fell down a flight of stairs on Jan. 28, 1942, while fire-watching and was admitted to Crumpsall Hospital in a semi-comatose condition. A moderate degree of concussion was present, the patient being drowsy but co-operative. There was bruising of the right pinna and mastoid region and a discharge of watery fluid from the right ear, which also contained some purulent discharge, suggesting the presence of chronic otitis media with a recent fracture of the base of the skull involving the middle ear and allowing the escape of cerebrospinal fluid. A swab taken from the ear showed diphtheroid organisms. Radiographs taken on the day of admission showed air in both lateral ventricles, which were symmetrically placed, and some scattered areas of air on the surface of the brain, probably in the subarachnoid space; the whole appearance was that of a normal encephalogram (*Fig.* 255). The patient made good progress up to the fourth day, making an apparently good recovery from concussion, although the cerebrospinal otorrhœa still persisted. On the fifth day he suddenly became semi-conscious, with a rise of temperature to 101° F. and marked tachycardia. The right pupil became dilated while the left remained normal, both reacting to light; the reflexes were all brisk and the plantar responses flexor. Neck rigidity was present and the condition strongly suggested the supervention of meningitis, which was confirmed by lumbar puncture, which showed slightly turbid, blood-stained fluid containing 730 white blood-cells per c.mm., mostly polymorphonuclear leucocytes and numerous Gram-positive bacilli on examination of the centrifuged deposit. On culture this fluid grew an organism identical with the diphtheroid obtained from the ear swab. The patient died within a few hours of the onset of meningitis, and post-mortem examination showed extensive acute purulent meningitis over the base of the brain and throughout the ventricular system. There was a fracture of the right middle fossa of the base of the skull extending into the middle ear, with a tear of the dura in this situation.

DISCUSSION

Traumatic pneumocephalus may be divided according to the situation of the air, into four types: extradural, subdural, intracerebral, and ventricular. The last type may be of two varieties: (*a*) where the air reaches the ventricle

by rupture of an intracerebral collection of air into the ventricle (the *pneumatocéle térébrant* of Worms, Didiée, and Grumbach[3]); and (*b*) where the air escapes from the affected air sinus through the dural tear into the subarachnoid space and

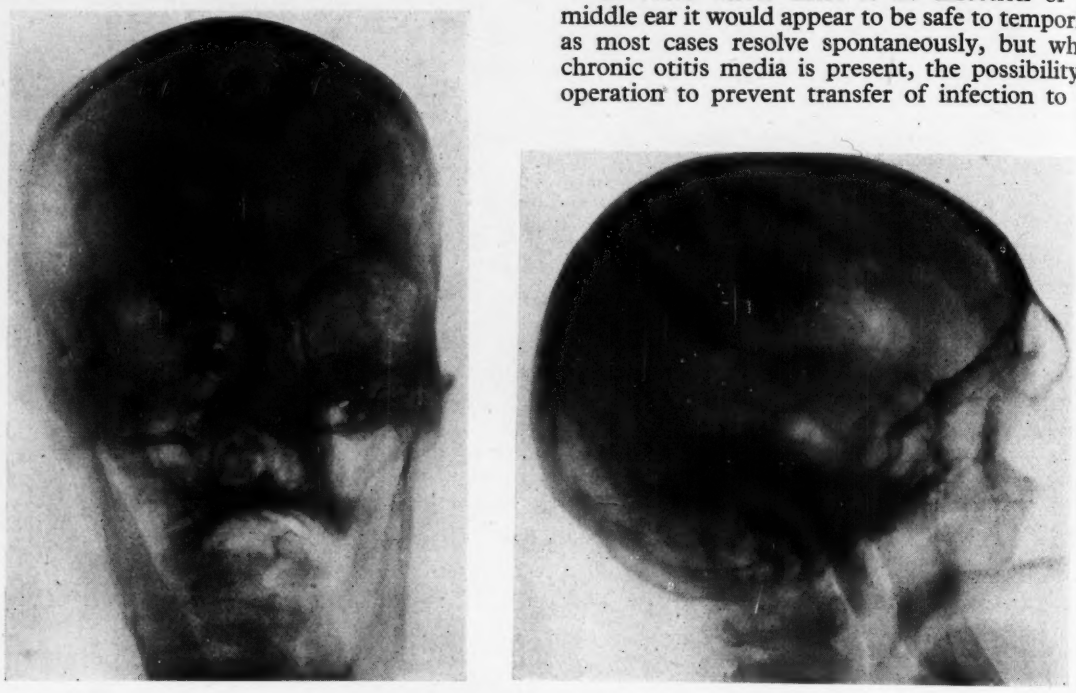

FIG. 255.—Radiographs showing air in the lateral ventricles and in scattered areas on the brain surface. A, Antero-posterior; B, Lateral.

reaches the ventricular system by the same route as does the air introduced for a diagnostic encephalogram, via the foramina of Lushka and Majendie in the fourth ventricle. In the majority of recorded cases of this latter variety the entry of air into the ventricles occurs immediately after the injury, and air is seen in the ventricles on the first radiograph taken. This occurrence seems to be rare in fractures involving the middle ear or mastoid sinus, and none of the English authors record it, though it was present in 6 of the 72 cases collected by Worms, Didiée, and Grumbach.[3] In all of these the patients recovered without operation, the air being absorbed in a few weeks.

The interest of the present case lies in the problem of the advisability of surgical intervention at the earliest moment to prevent the occurrence of meningitis—could he have been saved by immediate operation and closure of the dural tear? Most of the cases of pneumocephalus previously described have been intracerebral collections of air discovered some weeks or months after the original trauma, and operation has been carried out in a number of these, the most successful being those on the lines described by Dandy,[2] incising the aerocele and closing the communication with the air sinus concerned by suture of the dural rent or by means of a patch of fascia lata. It should be possible to perform this operation for tears of the dura over fractures involving the middle ear, though I can find no record of this having been done.

In cases where there is no infection of the middle ear it would appear to be safe to temporize, as most cases resolve spontaneously, but where chronic otitis media is present, the possibility of operation to prevent transfer of infection to the meninges must be considered.[9, 12] Dandy[13], in a recent publication, says that communication from the subarachnoid space to an infected mastoid sinus is almost invariably fatal, though Rand[14] considers that the dural tear should be closed at once and that meningitis should not necessarily deter an attempt to correct the pneumocephalus, as success depends largely on early recognition and prompt intervention. This is supported by Cairns,[9] while in a recently published work on head injuries, Rowbotham[15] states that in cases of fracture of the skull with cerebrospinal fluid from the ear, if chronic otitis media is present, a radical mastoidectomy should be carried out as soon as the patient's condition permits.

It would therefore appear that in cases where the sinus concerned is infected the patient's best chance would lie in immediate operative treatment, as the risk of operation is less than that of meningitis.

SUMMARY

A case of traumatic ventricular pneumocephalus resulting from a fracture of the skull involving an infected middle ear is described, and the advisability of immediate operative treatment in similar cases is discussed.

My thanks are due to Professor G. Jefferson for his encouragement and advice; and to Dr. W. A. Ramsay, Medical Superintendent of Crumpsall Hospital, Manchester, for his permission to publish this case.

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ACUTE DIVERTICULITIS OF THE JEJUNUM

BY GERALD H. C. OVENS, LONDON

DIVERTICULOSIS of the small intestine is an uncommon condition, but its importance as a cause of chronic dyspepsia, and occasionally of an acute surgical emergency, is becoming increasingly recognized. Kozinin and Jennings[1] recorded 219 cases up to the end of 1941. Most of these had been uncomplicated, many being discovered post mortem or found accidentally during operations for other conditions. Quite certainly the condition is more common than has previously been supposed. Rosedale and Lawrence,[2] employing special methods, found 4 cases in 300 autopsies.

Usually the diverticula give rise to no symptoms. If uncomplicated they may cause a particular type of dyspepsia (Edwards[3]) consisting of left upper abdominal discomfort or pain coming on shortly after meals, flatulence, and borborygmi; these symptoms may be associated with attacks of colic or diarrhœa, and sometimes with bleeding into the lumen of the bowel.

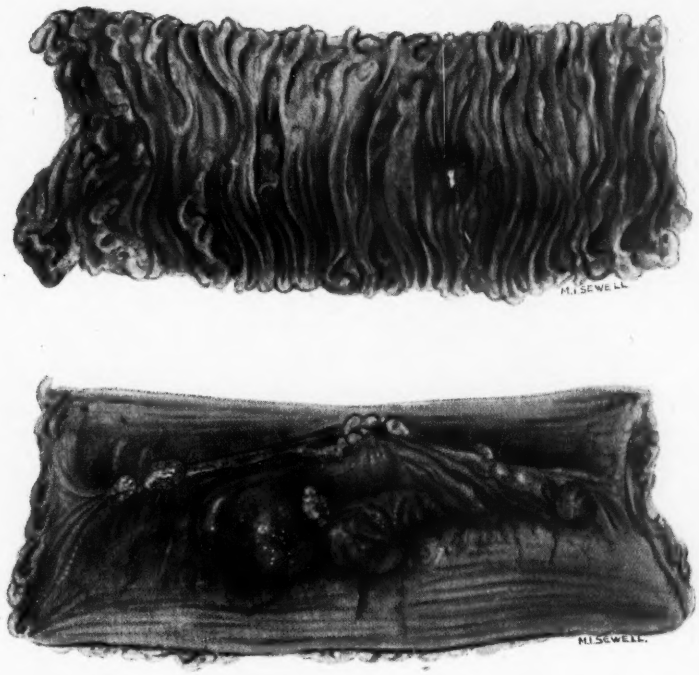

FIG. 256.—Resected portion of the bowel showing acutely inflamed diverticulum with surrounding peritonitis. Two uninfected diverticula are also seen. The openings of the latter are concealed under the folds of mucous membrane.

The acute complications of inflammation, obstruction, and torsion are much less common. This is due to the fluid nature and sterility of the contents of the jejunum. Gerster,[4] in an extensive review of the literature, collected 187

cases of diverticulosis of the small intestine reported up to May, 1938, but in only 12 of these was there acute inflammation or perforation. The following additional case of acute diverticulitis is here recorded.

CASE REPORT

HISTORY.—Mrs. N. R., aged 59, was admitted to St. Bernard's Emergency Hospital at 10.30 p.m. on May 4, 1941. Her general health had always been excellent, but for the last two or three years she had suffered from transient attacks of upper abdominal pain. These attacks would come on at night after she was in bed, were rather severe, but always went away if she took a " drop of hot water ". They were never bad enough to cause serious inconvenience.

FIG. 257.—Barium-meal radiograph taken a month after operation. One large and one small diverticulum may be seen filled with the barium.

Five nights previous to her admission, she had had one of these attacks, which had failed to clear up as usual, and had been succeeded by a dull aching pain in the epigastrium. She continued about her usual household duties for the next few days, but the aching persisted and she had also one or two attacks of intestinal colic. Fourteen hours before admission she vomited for the first time. After this the pain became more severe and more generalized.

Physical examination showed a very ill woman with signs of general peritonitis. A tentative diagnosis of perforated appendicitis was made.

AT OPERATION.—Under spinal anæsthesia. A diffuse generalized peritonitis was found and multiple diverticula of the jejunum were seen. These extended from the duodeno-jejunal junction for a distance of approximately 8 ft. down the bowel. The uppermost lay immediately beneath the transverse mesocolon, and all were situated along the mesenteric border. They varied in size from a pea to a golf-ball, and were far too numerous to justify a count. All were healthy except for one situated about 9 in. from the duodeno-jejunal junction. Here the bowel was wrapped round with inflamed omentum, and when this was separated, an acutely inflamed, unperforated diverticulum was seen. The unhealthy condition of the neighbouring part of the bowel rendered a simple diverticulectomy impossible, while the site of the highest diverticulum at the root of the mesocolon and the poor general condition of the patient prevented the complete resection of the whole of the affected part of the bowel. Some 8 in. of the jejunum, including the inflamed diverticulum, was removed (*Fig.* 256), an end-to-end anastomosis performed, sulphanilamide powder scattered into the peritoneal cavity, and the abdomen closed without drainage.

PROGRESS.—The post-operative course was uneventful, and after a barium meal which showed some of the remaining diverticula (*Fig.* 257), the patient was discharged on June 20, 1941. A month later she was re-admitted for a barium enema, but this showed no diverticula of the colon. When seen a year after her operation she was perfectly well, and had had no recurrence of her indigestion.

SUMMARY

1. Attention is drawn to the condition of diverticulosis of the jejunum as a cause of chronic dyspepsia and occasionally of an acute surgical emergency.

2. A successful case of resection for acute diverticulitis is recorded.

I have to thank the Medical Superintendent of St. Bernard's Emergency Hospital and Dr. W. Allen Daley, of the London County Council, for permission to publish this case.

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AIR ARTHROGRAPHY IN LESIONS OF THE SEMILUNAR CARTILAGES

BY C. H. CULLEN AND G. Q. CHANCE

THE principle of using contrast radiography in diagnosis of joint lesions is now well established as a useful adjunct to clinical examination.

Iodized oils or the uroselectan group present certain disadvantages. They are irritant and tend to throw a dense shadow which obscures fine detail. Injection of gases into the joint is a method which has been used by many workers.

Fullerton during the last war used a combination of air injection and radiography to delineate the extensions of the complicated synovial sacs of the knee. He was particularly interested from the viewpoint of the surgeon called on to operate for F.B.'s in this region. Quaintance[1] used this method in 50 patients, and quotes quite a long list of contributions to the literature from Baleming, Bernstein, Arens, Hamilton, and others, mainly of isolated cases. In his own series 24 came to operation; fracture of the cartilages was verified in 71 per cent of the cases, and decision to operate were largely based on clinical data.

Method.—The skin and capsule having been infiltrated with novocain, the suprapatellar pouch is punctured ½ in. above and lateral to the patella. Air is injected with a 20-c.c. syringe attached to the needle by a three-way adaptor. The air is filtered by covering the inlet with two or three layers of gauze swabs. Any fluid present is aspirated before injection. The amount of air used varies from 70 c.c. to 140 c.c. in different cases. Injection is continued till the patient feels his knee definitely tight and there is sufficient tension to blow back slowly the piston of the syringe. The needle is then withdrawn and the puncture sealed.

There are several minor difficulties which are easily overcome:—

1. The air may be injected into the extra-articular space. This is easily detected by increased resistance to injection and the appearance of crepitus on deep pressure. The needle must then be re-inserted.

2. Air may leak through the puncture on withdrawing the needle. This can be obviated by introducing the needle obliquely and by maintaining firm pressure on the puncture site with a finger for two or three minutes. (In one case with a large communication with an extra-articular bursa, filling of the joint was unsatisfactory and the radiograph consequently of poor

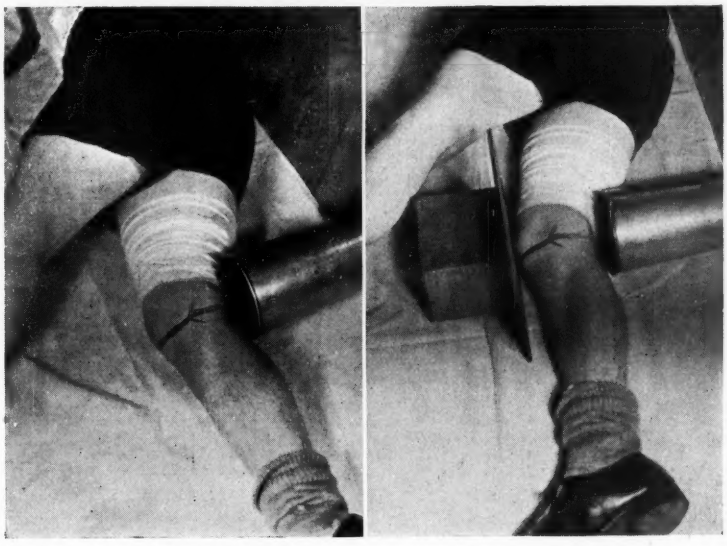

FIG. 258.—Photographs showing the position to be adopted.

displacement in 91 per cent. He came to the conclusion that the procedure is simple, harmless, and of definite value in diagnosis of knee-joint injury.

The principle has been applied by us to the examination of a series of patients admitted to hospital with a preliminary diagnosis of internal derangement of the knee-joint.

The investigation was undertaken in an attempt to correlate the radiological with the clinical and operative findings. Our diagnosis

quality. Repetition of the injection, using larger amounts of air, gave useful films). The air is not withdrawn at the end of the examination and does not appear to cause any discomfort. It is slowly absorbed over a period of four to five days

FIG. 259.—Sketch to illustrate positioning so that central ray (indicated by arrow) passes through the cartilage to be examined.

and gives time for further examinations if these are considered necessary. Three patients developed slightly turbid effusions. Two came to operation and the fluid was removed. One patient had an aspiration and the fluid did not recur.

No major reactions were noted in this series, but Kleinberg cited a case of non-fatal pulmonary embolus in 1927 following air injection.

At this stage the patient arrives in the X-ray department.

Bandaging.—In order to ensure that the air in the knee-joint is under pressure, a firm bandage is applied to the suprapatellar bursa starting from above.

Location of the Joint.—As the knee is slightly flexed by the bandage and somewhat distorted and swollen, it is essential that the joint space should be accurately localized. If the central beam does not pass through the joint space at exactly the right angle, there occurs an overlapping of the images of femur and tibia which greatly confuses the result. For this

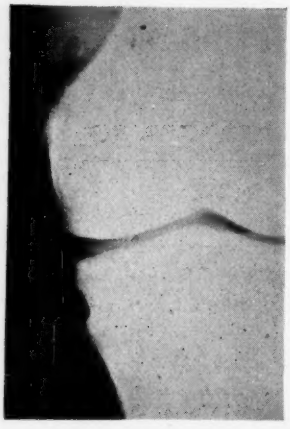

FIG. 260.—The normal knee. Cartilage is clearly seen between the femur and tibia. No air shadow below or in cartilage. The internal lateral ligament is not well demonstrated in this photo, but is usually well seen.

reason the joint space is very quickly screened and outlined with skin pencil immediately after bandaging. It is important that this should not be done before the bandaging as the latter alters the skin position considerably.

Radiography.—The tube is arranged so that its beam passes in a horizontal direction. It is never moved during the examination. When a new view is required, the position of the knee is altered. For accurate centreing I use a special cone about 30 in. in length, with an aperture of about 6 in. in diameter. The knee is arranged so that the central beam passes through the side of the knee in a glancing way (*Fig.* 258). The aim is, that the central beam should pass through the portion of the cartilage to be examined and through as little else as possible. Incidentally, the part of the knee being examined is always upwards, so as to ensure the maximum

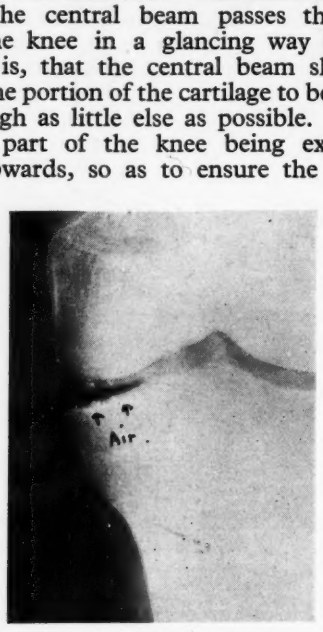

FIG. 261.—Separation of cartilage from tibia. Probably the earliest stage of injury. Air shadow is clearly seen between cartilage and tibia.

air filling. We add a rough sketch to illustrate this point (*Fig.* 259). The tangential nature of these views is of the utmost importance and makes the films more interpretable by elimination of confusion caused by superimposition of shadows.

Technique.—Using unscreened films without grid and working at 36 in. F.S.D., use 200 ma./sec. at 60 kv.

The most important advances in our technique have been the introduction of tangential views

FIG. 262.—Fracture of cartilage. Air is seen above and below the cartilage. A fracture is present in the cartilage, and the periphery of cartilage is displaced outwards from the joint. Film retouched, but still accurate. F, Fracture; C, Cartilage.

and the rendering of the contrast medium under some pressure. This pressure ensures that any space within the knee will be filled with air, and where no air shadow can be seen in the film one can reasonably presume that no such space exists.

Interpretation.—With the aid of a slight knowledge of anatomy and pathology, combined with common sense, we have endeavoured to work out the signs which might be considered pathological. The films were interpreted by one of us without any clinical knowledge of the cases and the opinion was checked, in nearly all cases, by the findings at operation. *Fig.* 260 shows the appearance of a normal knee.

That we have achieved quite a high percentage of accuracy in our first series has been somewhat of a surprise, if a pleasant one.

According to our present imperfect knowledge, we recognize roughly five types of findings. There are, of course, many intermediate types and combinations of these. These five types are :—

1. Separation of cartilage from lateral ligament.
2. Separation of cartilage from tibia (*Fig.* 261).
3. Fracture of cartilage (*Fig.* 262).
4. Fracture of cartilage with displacement of fragment.
5. Fragmentation, reaching in cases almost complete disappearance (*Figs.* 263–265).

The series under review consists of our first 32 cases. On clinical grounds alone the diagnosis was as follows :—

Definite cartilage lesion	12
Doubtful „ „ (probably positive)	5
„ „ „ („ negative)	5
No evidence of cartilage lesion	10

Twenty-two cases came to operation.

FIG. 263.—Four illustrations of the stage of fragmentation. The two upper films were touched up, the lower untouched. Displacement of the main body of cartilage (C) still attached to ligament. Inner rim of cartilage (L.B.) forms a loose body—which sometimes disappears later. A, Air.

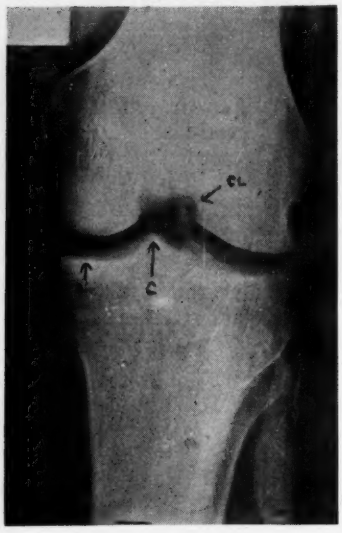

FIG. 264.—Inner fragment of cartilage turned into intercondylar space. Confirmed by operation. Space for medial cartilage entirely filled by air. Compare with external cartilage. C, Cartilage; C.L., Cruciate ligament.

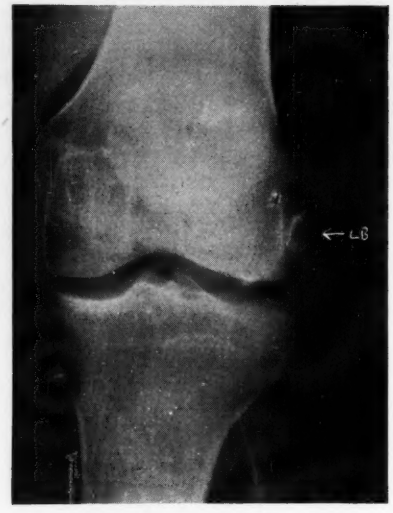

FIG. 265.—This case had been to many clinics and was always found negative. Our ordinary films were again negative, but on air arthrography a loose body showed up against the air-filled bursal sac. The loose body was removed by operation.

TOTAL CASES OPERATED UFON

NO. OF CASES	PATHOLOGY	AIR ARTHROGRAM
9	Typical bucket-handle fracture	Positive in all
4	Internal semilunar cartilage almost absent + erosion medial condyle	Positive in all. Erosion seen in one
2	Simple erosion	1 Negative; 1 Positive
1	Negative	Loose body seen later on arthrogram
2	Mobile internal semilunar cartilage	Positive in 1; doubtful in 1
1	Longitudinal split, I.S.C.	Positive
1	Transverse tear, I.S.C. in anterior two-thirds	Positive
1	External cartilage cyst	Negative
1	Mobile external semilunar cartilage	Negative

DOUBTFUL CARTILAGE LESIONS
(*probably positive clinically*)

NO. OF CASES	PATHOLOGY	AIR ARTHROGRAM
2	Bucket-handle fracture	Positive in both
1	Erosion medial condyle	Negative
1	Mobile external semilunar cartilage	Negative
1	Negative	Loose body seen later

DOUBTFUL CARTILAGE LESIONS
(*probably negative clinically*)

NO. OF CASES	PATHOLOGY	AIR ARTHROGRAM
4	Bucket-handle fracture	Positive in all
1	Mobile internal semilunar cartilage	Positive

Ten cases did not come to operation. In only one was a definite diagnosis of osteo-arthritis made clinically and radiologically. A definite diagnosis was not possible in the others. The arthrogram was normal in 4, inconclusive in 4, and in 1 suggested internal semilunar cartilage lesion (not followed).

All of these patients had a course of physiotherapy and severe P.T.; in none of them did the knee in question cause further trouble, and they were subsequently discharged. In these the presence of a lesion or its absence was not proven by operation, but their subsequent course makes it likely that the diagnosis was correct.

COMMENTARY

An air arthrogram showed definite lesions in 18 of the 22 patients whose knee-joints were explored. The other 4 were negative on X-ray examination, but the following lesions were found :—

1 case external cartilage cyst (clinically obvious)
1 „ erosion medial condyle
1 „ mobile external semilunar cartilage and hypertrophied infrapatellar pad
1 „ completely negative except for small erosion on medial condyle. (On reconsidering this case either a loose or pedunculated body was seen in the films.) *Case* 2.

Seventeen patients would have had operation on clinical grounds; the remaining five, in all of which definite lesions were found at operation, would probably have been rejected on these standards.

In one case the arthrogram showed displacement of the medial cartilage, but at operation this was not considered sufficiently severe to warrant its removal.

In the clinically negative cases (10) arthrography proved the cartilages to be normal in four. In one there was evidence of cartilage injury, and I think in spite of negative clinical findings this man should have had his knee explored. In the remaining five air arthrography indicated pathology, and operation confirmed X-ray findings.

Recently, an attempt was made to X-ray an air-filled knee by planigraphy, taking sections at ½-in. intervals. The case was clinically positive, the X-ray finding indicated that it was not pathological, and at operation the knee was found to be normal. To evaluate this method, however, it will be necessary to do a further series of cases.

CONCLUSION

Air arthrography is a simple painless method of examination. By its aid the semilunar cartilages in the knee can be visualized, and definite help given to the clinician in the diagnosis of difficult cases. There is a high percentage correlation between the clinical and radiological diagnosis. We believe that the method is worthy of extended trial, and radiographs taken in conjunction with the clinical picture can increase the percentage of positive cases coming to operation.

REFERENCE

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EXPERIMENTAL SURGERY

EXPERIMENTAL BRAIN ABSCESSES IN THE RABBIT

BY MURRAY A. FALCONER, ALLAN M. McFARLAN, AND DOROTHY S. RUSSELL*

FROM THE NUFFIELD DEPARTMENT OF SURGERY AND THE EMERGENCY PUBLIC HEALTH LABORATORY SERVICE, OXFORD

THE study here reported was undertaken in order to observe stages in the formation of brain abscesses resulting from infection with different organisms. In particular we wished to analyse the process of encapsulation, to observe the time relationships of the different stages, and to ascertain whether the process varies with different infecting organisms. This last-mentioned quest was prompted by the assumption which recurs in clinical reports (Neumann, 1909; Alpers, 1939; Grant, 1941) that abscesses caused by anaerobic and Gram-negative organisms are either poorly encapsulated or entirely defective in this respect. We have also made some observations on the effects of thorotrast when introduced into brain abscesses and on the effects of systemic sulphonamide therapy in streptococcal infections. The technique used and our preliminary observations were given in a short communication to the Society of British Neurological Surgeons (Russell, Falconer, and McFarlan, 1941).

EXPERIMENTAL METHODS

1. Operative.—Adult rabbits were used. In the majority of experiments the inoculation of the brain was carried out in two stages. The operative procedure employed in the first stage was similar to that used by us for other investigations (Russell and Falconer, 1941). The animals were anæsthetized by the intravenous injection of nembutal solution supplemented when necessary by the inhalation of ether. Under aseptic conditions a midline incision was made through the scalp. A triangular area of bone (averaging 1·5 cm. long by 1 cm. wide) was removed from over the centre of the left cerebral hemisphere (*Fig.* 266) and the brain exposed by

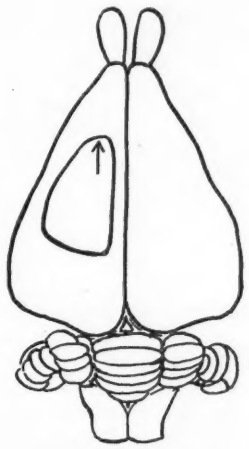

FIG. 266.—Diagram to show site of operation. Arrow indicates direction in which inoculation is made (*see text*).

opening and reflecting the dura. The surface of the brain was not disturbed. The scalp incision was then sutured and the wound allowed to heal. The second stage was undertaken ten or more days later, when healing had occurred. Under ether anæsthesia the brain was inoculated through the skin by injecting 0·1 c.c. of 2 per cent nutrient agar containing the selected strain of organisms in suspension (*see below*). In order to avoid the ventricle the needle was inserted at the anterior angle of the decompression and directed forwards so that the injection was made into the subcortical white matter.

* Working for the Medical Research Council.

In two small groups of animals we employed simpler methods of inoculation, but the results were not so satisfactory. In one group we injected 0·1 to 0·15 c.c. of a 24-hour broth culture of organisms (*Str. pyogenes*, 6 experiments; *Staph. aureus*, 4 experiments) instead of a suspension of organisms in agar. Although the number of organisms introduced was about ten times that used when the culture was diluted with agar, the percentage of infections produced by this technique was less than with the standard method. (Infections were produced in 5 out of 10 as compared with 65 out of the other 79 experiments made with these organisms; of the 5 infections resulting from the injections without agar only 3 examples were abscesses and these were associated with pyocephalus and meningitis.) In the other group the brain was inoculated in one stage by making a small burr hole over the left hemisphere and injecting 0·1 c.c. of a suspension of organisms in agar (*Staph. aureus*, 6 experiments) through the intact dura into the subcortical white matter at the usual site. After each injection we noted a slight reflux of agar immediately following withdrawal of the needle, and in half of the experiments we subsequently found that the subdural space had been infected as well as the cerebrum.

The standard method of inoculation used in the majority of our experiments has therefore certain advantages over these simpler methods. It entails less risk of a spreading leptomeningitis and pachymeningitis because the injection is made through an area where the subarachnoid and subdural spaces have been sealed off during healing of the decompression wound. The use of agar in the inoculum appears to favour the establishment of an infection. Finally, the provision of a bony decompression prevents marked rises in intracranial pressure by enabling the inflamed cerebral tissues to expand; a subsidiary advantage lies in the fact that the bulging of tissues through the decompression frequently enables the observer to tell whether the inoculation has been successful or not. The use of agar was suggested to us by the late Dr. W. M. Scott. We subsequently found that Groff (1934) had also employed agar and a similar two-stage technique, though with a smaller bony opening.

2. Bacteriological.—The organisms used were all derived from human sources (*Table I*). They were prepared for intracerebral inoculation by plating out the recently isolated or rabbit-passaged strain and subculturing a colony into broth or serum broth. After incubation for 24 hours (48 hours for the anaerobes) the culture was shaken and one or two drops, according to the turbidity, transferred in a Pasteur pipette to 1 c.c. of melted 2 per cent nutrient agar at about 40° C. The organisms were mixed with the agar and 0·1 c.c. withdrawn into a syringe for inoculation.

Cultures were made at autopsy from the brain abscesses and the heart blood in certain animals as described below (p. 254).

3. Pathological.—Most of the animals were killed by the inhalation of chloroform, and the tissues were at once perfused through a cannula inserted into the aorta with warm isotonic saline followed by 10 per cent formaldehyde. A few animals were anæsthetized with chloroform and killed by bleeding, so that cultures could be made of the cerebral abscess; these animals and those which were found dead were not perfused. In all animals the brain was then removed, and after being hardened in formaldehyde was divided into coronal slices about 2 mm. thick.

Selected coronal slices were embedded in paraffin wax and sections were stained with Ehrlich's hæmatoxylin and eosin, Weigert's iron hæmatoxylin and

Table I.—Organisms Used for Inoculation

Organism	Source	No. of Experiments	Remarks
Str. pyogenes	Brain abscess	42	Group A. Type V
Staph. aureus	1. Brain abscess	38	All coagulase-positive
	2. Brain abscess	2	
	3. Brain abscess	5	
	4. Wood strain	2	
Staph. albus	Nose swab	2	Coagulase-negative
Pneumococcus	1. Empyema	4	Type I
	2. Brain abscess	2	Type VII
	3. Throat swab	3	Type XIX
Proteus vulgaris	Brain abscess	7	
Bact. Friedländeri	Meningitis	2	
Anaerobic streptococci	1. Brain abscess	8	Obligate anaerobe. Isolated in pure culture
	2. Brain abscess	2	Obligate anaerobe. Isolated with fusiform bacilli
	3. Brain abscess	2	Micro-aerophilic. Isolated in in pure culture
*Fusiform bacilli	1. Brain abscess	2	Isolated with anaerobic streptococci
	2. Brain abscess	7	Isolated in pure culture

* The fusiform bacilli were Gram-negative, non-sporing, obligate anaerobes. They grew well on blood-agar and in serum broth, produced a foul odour in cooked meat, and showed coccobacillary and filamentous forms with irregular swellings in films from cultures. In peptone-water sugars acid without gas was produced in glucose, maltose, lactose, sucrose, and salicin. Mannite was not acidified.

van Gieson's mixture, phosphotungstic-acid hæmatoxylin, and Gram's stain for bacteria. Other slices were embedded in gelatine and frozen sections were impregnated with silver carbonate for reticulum fibres and also for microglia, using Penfield's modification.

RESULTS

1. Clinical.—The establishment of an intracranial infection was usually indicated by some degree of bulging of the decompression during the first few days after the inoculation. The more severe the infection the greater was the degree of bulging (*Fig.* 267). In the most acute infections systemic symptoms were also prominent; the animal would become quiet and refuse

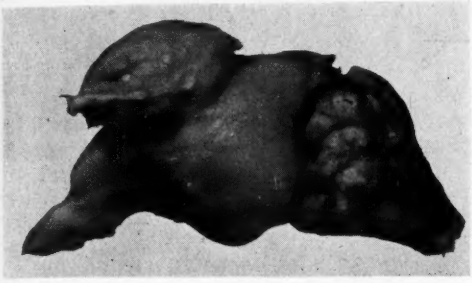

FIG. 267.—Left lateral view of rabbit's brain to show a large hernia 11 days after inoculation with hæmolytic streptococci. An abscess beneath the hernia has ruptured into the lateral ventricle, producing terminal meningitis.

its food, and later display irritability progressing to rigidity, opisthotonos, coma, and death within 48 hours. In less acute infections the clinical course was less rapid; in experiments which terminated fatally death usually occurred within the first week, but, if the animal were going to survive, the bulging of the decompression subsided and the systemic symptoms disappeared within a few days. Subsequently such animals usually maintained good health, with increase of body-weight, until they were killed. In the milder infections systemic symptoms were often absent. In animals inoculated with pneumococci, *Str. pyogenes*, or *Staph. aureus*, we came to rely upon the early observation of bulging of the decompression as evidence of an established infection. On the other hand, with organisms such as the anaerobic streptococcus, *Proteus vulgaris*, and fusiform bacilli we found that swelling of the brain was often less pronounced or even absent, and there was no systemic disturbance even though an abscess of considerable size might later be revealed at autopsy.

2. Pathological.—Altogether 130 animals were inoculated, as shown in *Tables I* and *II*. A total of 63 abscesses in various stages was obtained, of which 10 were confined to the meninges. In 35 others there was a diffuse inflammatory reaction within the cranium without evidence of localization at the site of inoculation: these are separated into leptomeningitis (7), and a spreading encephalitis, with focal or general leptomeningitis and usually pyocephalus. For convenience the term 'meningo-encephalitis' is used for this group which comprises 28 animals. No evidence of infection was found in 32 animals.

Coincidental Pathological Findings.—As reported earlier (Russell and Falconer, 1941), the first stage of the operation performed in these experiments is often followed by a variable degree of herniation of the brain which subsides within the following week. Hæmorrhage and necrosis in the white matter at the base of the hernia lead to the formation either of a traumatic cyst or scar. These effects are to be reported in a separate paper. In the present investigation we attempted to avoid this complication by setting aside those animals which developed a palpable hernia after the first stage. Nevertheless histological evidence of cyst formation was found in some of those inoculated, and 7 of these showed involvement of the cyst by organisms. Such examples have been discounted in the histological descriptions which follow.

Infection by the *Encephalitozoon cuniculi* was found in 4 of the rabbits which had developed a cerebral abscess, and in 1 which had a meningeal abscess. While there was no evidence that the coincidental infection modified the inflammatory reaction about such abscesses, these examples also have been left out of consideration in the following descriptions.

a. MENINGITIS.—The 7 animals in this group were infected with *Str. pyogenes* in 6 and pneumococci in 1. The streptococcal meningitis was acute in 3 animals dying within 48 hours of inoculation, and chronic in 3 which were killed from 9 to 21 days after inoculation. The pneumococcal example was an acute purulent meningitis, the animal dying within 48 hours of the inoculation. Histologically these animals showed no special features meriting a more detailed description.

b. MENINGO-ENCEPHALITIS.—There were 28 animals in this group. Ten were infected with *Str. pyogenes*, 8 with pneumococci, 8 with staphylococci, 1 with Friedländer's bacillus, and 1 with anaerobic streptococci. None survived for longer than 8 days and the majority (18) died within 48 hours.

Macroscopically these animals showed severe diffuse œdema of the cerebrum at the site of inoculation, with herniation of the brain through the decompression opening. On section a ragged cavity containing thin pus was often disclosed in the depths of the hernia, but with no evidence of demarcation of the inflammatory reaction in its walls. Softening of the tissues, often accompanied by small hæmorrhages, extended to the lateral ventricle and was associated with pyocephalus in 20 examples. The pyocephalus was sometimes conspicuous. In a few animals there was diffuse swelling, softening, and hæmorrhage, without cavitation of the cerebral tissues forming the hernia.

Microscopically there is severe necrosis of the cerebral tissues at the site of inoculation, extending as irregular tongues through the white matter and into the deeper layers of the cortex. This necrotic tissue contains numerous but unevenly

distributed organisms, and is devoid of inflammatory cellular reaction except where groups or undulating zones of leucocytes have become included in the necrosis. Well-preserved leucocytes are generally abundant about the periphery of the necrotic areas, especially where these

FIG. 268.—Œdema, necrosis, and diffuse purulent encephalitis extending into lateral ventricle (V) 4 days after inoculation with hæmolytic streptococci. (Hæmatoxylin and eosin.) (× 36.)

encroach on the cortex. In the white matter, however, and particularly towards the ventricle, the necrosis is often devoid of any limiting inflammatory reaction. In this way the ependyma may be involved, with a subsequent heavy infection of the ventricular system (*Fig.* 268). The degree to which hæmorrhage and the escape of fibrin from the vessels play a part in the reaction varies not only amongst the different types of infecting organisms, but individually amongst the animals infected with any one type of organism. The organisms are most abundant in the areas of necrosis, but a few are usually found beyond the periphery of such areas. Here they appear to be encroaching upon the brain tissue in a diffuse manner and independently of the blood-vessels. Such tissue shows severe œdema and degeneration. Quite often organisms can be demonstrated within the cytoplasm of mononuclear amœboid cells which, from a study of suitably impregnated sections, appear to be microglial cells. These cells become activated and multiply in the neighbourhood of the infected tissues within 24 to 48 hours. Pyocephalus is invariably accompanied by an acute diffuse purulent or hæmorrhagico-purulent leptomeningitis which is more pronounced at the base of the brain than elsewhere. In 7 examples acute meningitis was limited to the cerebral convexities in the neighbourhood of the decompression. The single example in which the infecting organism was an anaerobic streptococcus is included in this group although the histological picture was different. In this animal, which was killed 8 days after inoculation, the meninges and superficial layers of the cortex at the site of infection showed a subacute inflammatory infiltration; the reaction was strictly focal and evidently represented a stage of healing.

c. ABSCESS.—As shown in *Table II* a total of 63 abscesses was obtained. Of these, 10 occupied the meninges at the site of inoculation, while the remaining 53 were cerebral.

i. *Meningeal Abscess.*—The meningeal abscesses were due to hæmolytic streptococci in 5, anaerobic streptococci in 2, and *Staph. aureus* in 3 cases. They resulted from direct inoculation of the meninges, the underlying cerebrum being usually unaffected except for inflammatory infiltration of the adjacent superficial layers of the cortex. Four of the animals (2 with hæmolytic streptococci and 2 with staphylococci) were killed at early stages varying from 2 to 5 days. One infected with anaerobic streptococci was killed at 10 days; the remainder were killed at from 3 to 8 weeks. The abscesses were visible to the naked eye as a circumscribed plaque of cheesy white exudate overlying the cortex near the site of inoculation and often deforming the adjacent cerebrum. In the

Table II.—RESULTS OF INOCULATION

ORGANISM	NO. OF EXPERIMENTS	NO INFECTION	MENINGITIS	MENINGO-ENCEPHALITIS	ABSCESS
Streptococci:					
(*a*) Hæmolytic	42	9	6	10	17
(*b*) Anaerobic	12	3	0	1	8
Staphylococci:					
(*a*) *Aureus*	47	10	0	8	29
(*b*) *Albus*	2	2	0	0	0
Pneumococci	9	0	1	8	0
Proteus vulgaris	7	3	0	0	4
Fusiform bacilli	9	4	0	0	5
Bact. Friedländeri	2	1	0	1	0
Totals	130	32	7	28	63

early stages the pus also occupied the adjacent subdural space; in the later stages (from 10 days onwards) the dura was bound down by fibrous adhesions to the surface of the abscess and the surrounding brain.

Microscopically the abscess at an early stage is composed of a degenerating mass of leucocytes surrounding the agar, in which numerous colonies of organisms are present. An excess of large mononuclear cells occupies the adjacent pia, both between the abscess and the cortex and at the lateral margins of the abscess. The subjacent superficial layers of the cortex are diffusely infiltrated with proliferating microglial cells and small lymphocytes. There is cuffing with lymphocytes of the superficial segments of the perforating vessels.

In the later stages the picture is altered through the appearance, at the periphery of the abscess, of a substantial zone composed of large mononuclear cells, fibroblasts, and foreign-body giant-cells embedded in young fibrous tissue. The giant-cells appear to be a response to the presence of the agar. In the oldest lesion (2 months) the cocci present are few and show little affinity for Gram's stain.

ii. *Cerebral Abscess.*—The 53 examples in this group were due to hæmolytic streptococci in 12, *Staph. aureus* in 26, anaerobic streptococci in 6, *P. vulgaris* in 4, and fusiform bacilli in 5. In 8 of the staphylococcal abscesses thorotrast had also been injected and a description of these is reserved till later (p. 256). The majority of our abscesses were obtained with hæmolytic streptococci and *Staph. aureus*. The following account is based mainly on the staphylococcal examples because early stages were observed in this group alone. A consideration of the whole series showed, however, that the inflammatory reactions obtained with the different organisms conformed to the pattern indicated in our general description—such differences as exist will be dealt with in a later section. The figures given in brackets refer to the staphylococcal group.

α. *Stages of evolution*: After 12 *hours* (2 examples) the macroscopical and microscopical appearances at the site of inoculation resembled those found in the rapidly fatal examples of purulent meningo-encephalitis, except that the area of necrosis was smaller and more circumscribed, being separated at most points from the adjacent tissues by a well-defined zone of leucocytes. Organisms were not found outside the central necrosis.

At 24 *hours* (1 example) a central well-defined core of pus containing irregular masses of agar had replaced the necrosis. A poorly demarcated narrow zone of necrosis, however, separated this central core from the adjacent cortex. Where the abscess abutted on white matter this zone was absent; instead, the border was blurred through the uneven infiltration of the tissues with polymorphonuclear leucocytes. A similar but sparser infiltration was present also in the cortex peripheral to the zone of necrosis, and in the zone itself. Numerous cocci, often in colonies, were present within the central core, but did not penetrate beyond its confines.

At 48 *hours* (2 examples) the appearances were similar, but the peripheral zone of necrosis was now more clearly defined and completely enclosed the central purulent core. Leucocytes were very sparse outside this. Early mobilization of microglial cells was evident in the adjacent nervous tissues, and a few of these cells, in the amœboid phase, occupied the zone of necrosis. As before, cocci were confined to the central pus.

At 3 *days* (2 examples) progress was shown by a greater degree of microglial infiltration outside the zone of necrosis and in one specimen a moderate number of these cells had become spherical and contained small granules of sudanophil lipoid. The adventitial sheaths of a few of the adjacent perforating vessels were now infiltrated with small lymphocytes, a few large mononuclear cells, and polymorphonuclear leucocytes. Corresponding to this infiltration there was an apparent increase of reticulum fibres in the walls of the vessels, probably due to splitting of the collagen by cells. No reticulum fibres were demonstrated outside the vessels. The adjacent nervous tissues were still œdematous and many neurones showed stages of chromatolysis and pyknosis of their nuclei.

At 1 *week* (6 examples; *Fig.* 284; streptococcal example, *Figs.* 269–271) the zone of necrosis had usually disappeared from the dorso-lateral border of the abscess towards the meninges, but was well defined elsewhere, especially in the white matter. Microglial reaction, with the formation of foam-cells containing lipoid, was still active outside this zone. Cocci were confined to the central pus and were especially abundant in the residual masses of agar. They rarely penetrated into the zone of necrosis, where they usually occupied the cytoplasm of the few foam-cells that had migrated centripetally. There was an appreciable proliferation of the blood-vessels in the adjacent cortex, but not in the white matter. Infiltration of the adventitial sheaths of these vessels was generally conspicuous and was accompanied by delicate intervascular reticulum fibres forming a narrow layer just beyond the zone of necrosis or its usual site. These fibres appeared first at the more peripheral parts of the cortex and diminished in numbers and density towards the white matter, where none was found. One abscess, which was completely intracortical, was invested with reticulum fibres on all aspects. Infiltration with lymphocytes and plasma cells was as a rule a conspicuous feature in the neighbourhood of the reticulum fibres, but varied a good deal in the different specimens.

At 2 *weeks* (2 examples) the appearances were essentially similar, except that in one example, which occupied white matter as well as cortex, the investment of the abscess by reticulum fibres was complete.

At 3 *weeks* (3 examples, *Fig.* 272) the wall of the abscess had now become organized into definite layers. The zone of necrosis had completely disappeared, except in the instance where it still occupied a short segment adjacent to the ventricle. Here encapsulation was in

abeyance. Elsewhere in all three specimens the central pus was enclosed first by a layer of closely packed foam-cells stuffed with sudanophil granules and separated by a delicate framework of reticulum fibres. Outside this came a middle layer of small lymphocytes, plasma cells, occasional spindle fibroblasts, and sparser unevenly distributed groups of more foam-cells. The reticulum fibres were circumferentially arranged and of coarser calibre than in the inner layer; fine collagen fibres could also be detected with van Gieson's stain. Foreign-body giant-cells were present in this layer and they occasionally occupied the inner layer as well. Finally, an ill-defined outer zone could be distinguished in which reticulum fibres became fewer and were associated with a sparse inflammatory infiltration with lymphocytes, plasma cells, and reacting microglial cells. Proliferation of fibrillary astrocytes was also evident in this zone; it was always feebler in the white matter than in the grey. The blood-vessels in this zone and for a variable distance beyond it continued to show cuffs of inflammatory cells and an increase of reticulum in their walls.

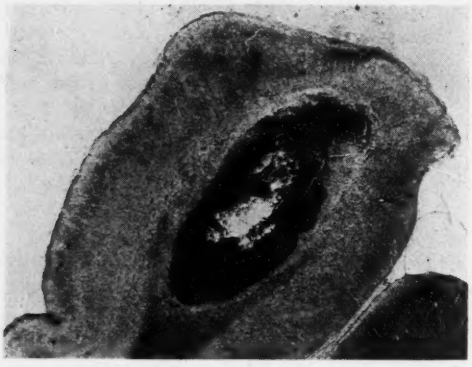

FIG. 269.—Abscess 1 week after inoculation with hæmolytic streptococci. (Phosphotungstic-acid hæmatoxylin.) (× 9.)

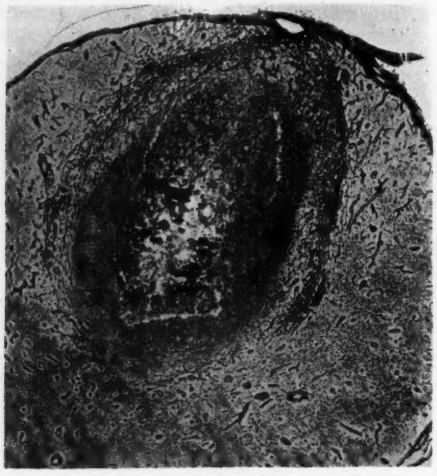

FIG. 270.—Same specimen as in *Fig.* 269 to show development of reticulum fibres about intracortical part of the abscess. (Silver carbonate.) (× 16.)

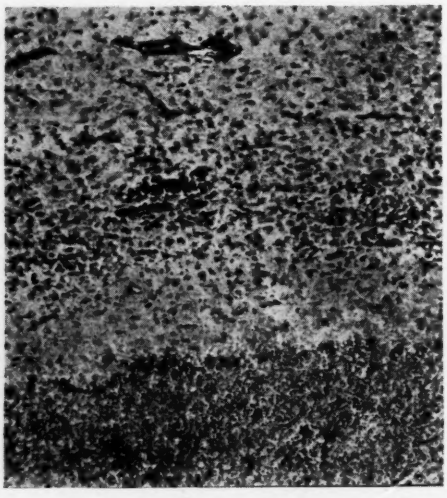

FIG. 271.—Same as *Fig.* 269, showing zone of necrosis at border of abscess and inflammatory reaction in cortex above this. (Hæmatoxylin and eosin.) (× 85.)

FIG. 272.—Abscess 3 weeks after inoculation with staphylococci. (Silver carbonate for reticulum fibres.) (× 15.)

From 3 *weeks onwards* no further evolutionary changes appeared in the abscess wall in the staphylococcal series, except consolidation of the reticulum and collagen fibres to form a dense felted layer at the periphery of the middle layer of the capsule, i.e., internal to the gliosis. This was well illustrated at 3 *months* in an abscess measuring about 0·5 cm. diameter and yielding a pure growth of *Staph. aureus* on culture. In an

abscess of the same size due to the hæmolytic streptococcus the appearances were similar, though the collagenous tissue was less robust

FIG. 273.—Abscess 3 months after inoculation with hæmolytic streptococci, showing well-formed capsule on all aspects. (Weigert's iron hæmatoxylin and van Gieson.) (× 8.)

β. Stages of resolution: After 3 months our observations were confined to the series obtained with the hæmolytic streptococcus. Two animals which had appeared on clinical grounds to have established foci of infection after inoculation were killed 6 months later. In both the abscess was represented by a relatively small oval area enclosed by a thin zone of delicate collagenous fibres and occupied either by cholesterin-crystal clefts bordered by foreign-body giant-cells, or by sparse foam-cells and small lymphocytes. No organisms were seen, and cultures taken from one were sterile. Two further examples of resolving abscesses were detected at the 2-month period (one due to hæmolytic streptococci and one to fusiform bacilli) and a third example at 2 weeks (fusiform bacilli). These represented earlier stages in the process of resolution. In them a small central core of pus was still present, but this area had been greatly reduced by the encroachment upon it of foam-cells and reticulum fibres from without. The inner layer of foam-cells was thus unusually deep. Fusiform bacilli were, however, still living and cultured from the 2-months-old abscess; they were not obtained from the 2-weeks-old specimen in the same group. The streptococcal example was not cultured, but a few

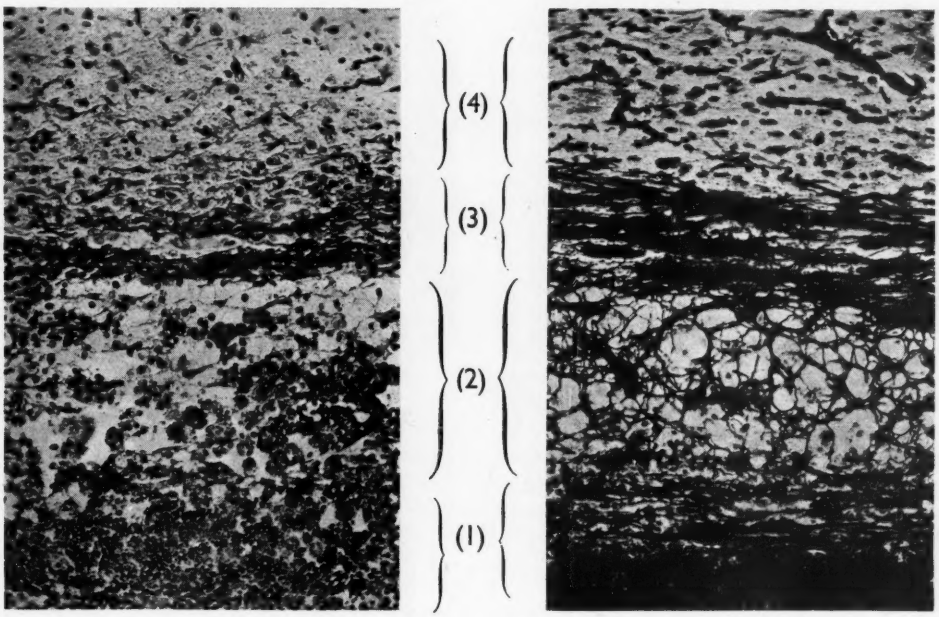

FIG. 274.—Same as in *Fig.* 273, showing (1) pus, (2) layer of fat-granule cells, (3) layer of circumferential collagen fibres and lymphocytes, and (4) glial reaction in adjacent cortex. (× 144.)

FIG. 275.—Cf. *Fig.* 274. Corresponding part of wall stained for reticulum fibres. (× 144.)

(*Figs.* 273–275). An earlier specimen at 1 month, due also to the hæmolytic streptococcus (*Figs.* 276–279), showed a capsule of less density than did most of the staphylococcal specimens at 3 weeks, or specimens at 4 weeks due to the anaerobic streptococcus (*Fig.* 280) and fusiform bacilli (*Fig.* 281).

groups of cocci of degenerate appearance were identified in sections.

γ. Loculation of abscesses: Three examples in our series showed loculation of the abscess. Two of these were due to hæmolytic streptococci and were obtained at 2 and 4 weeks respectively; the third was a staphylococcal abscess of 2 weeks'

duration. In all three the secondary abscess was situated in the region of the external capsule on the ventrolateral aspect of the main abscess.

FIG. 276.—Abscess 4 weeks after inoculation with hæmolytic streptococci, showing secondary loculus on ventrolateral aspect of main abscess. Note rarefaction of wall of abscess near ventricle. (Weigert's iron hæmatoxylin and van Gieson.) (× 8·4.)

FIG. 277.—Same specimen as in *Fig.* 276, stained for reticulum fibres. (× 8·4.)

FIG. 278.—Same as in *Fig.* 276, showing compact wall of abscess in cortex. (Hæmatoxylin and eosin.) (× 38.)

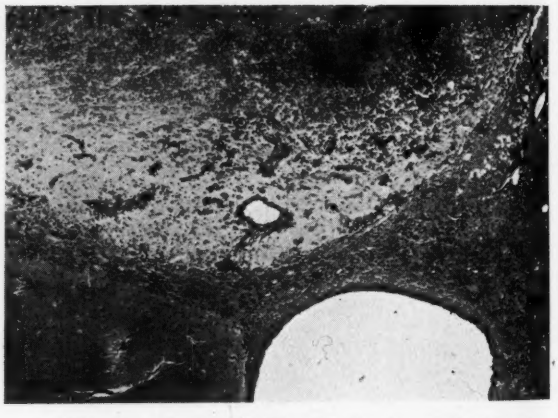

FIG. 279.—Same as in *Fig.* 276, showing rarefaction of tissues forming wall of abscess near ventricle. (Hæmatoxylin and eosin.) (× 38.)

At 2 weeks there was no clear histological evidence that the secondary loculus was of more recent origin than the primary abscess, but in the month-old example (*Fig.* 276) the stage of encapsulation reached in the secondary focus was less advanced than in the primary.

VARIATIONS ATTRIBUTABLE TO DIFFERENT TYPES OF INFECTING ORGANISM

A conspicuous feature in our experiments was the failure to obtain a localized purulent infection with pneumococci. Three types of this organism were tested and each produced a rapidly fatal meningo-encephalitis or meningitis in all animals inoculated.

With both hæmolytic streptococci and *Staph. aureus* a negative result was obtained in from 20 to 25 per cent of experiments; but while an acute spreading infection occurred in about 31 per cent of the streptococcal, it accounted for only 17 per cent of the staphylococcal infections (*Table II*). In other words, a larger proportion of the infections became stabilized to form abscesses in the staphylococcal group than in the streptococcal. This tendency to localization was still more evident with anaerobic streptococci, in which only 1 out of 12 animals showed a diffuse reaction, which was of a subacute or chronic type; and with *P. vulgaris* and fusiform bacilli, in which all the reactions obtained were localized. It may be permissible, therefore, to arrange these organisms in the following order of pathogenicity for the rabbit when inoculated intracerebrally: pneumococcus, hæmolytic streptococci, *Staph. aureus*, anaerobic streptococci, *P. vulgaris*, and fusiform bacilli.

A consideration of the stages of evolution observed in the cerebral abscesses in these different groups goes some way in support of this impression. In giving a general description of the process the staphylococcal group was taken as a pattern. The streptococcal group displayed similar features, but encapsulation was somewhat slower. Thus reticulum fibres were limited to a segment of the periphery in a specimen of 3 weeks' duration and, though in a 4-weeks' specimen the reticulum was complete, a segment of the zone of necrosis still persisted near the ventricle (*Figs.* 278, 279). On the other hand, with anaerobic streptococci, *P. vulgaris*, and fusiform bacilli the organization of the abscess wall was fully as advanced as with the staphylococcus, especially in the fusiformis group, where the reticulum zone was complete and the wall already showed a laminated structure at 2 weeks.

FIG. 280.—Abscess 4 weeks after inoculation with anaerobic streptococci, showing complete encapsulation. (Silver carbonate for reticulum.) (× 12.)

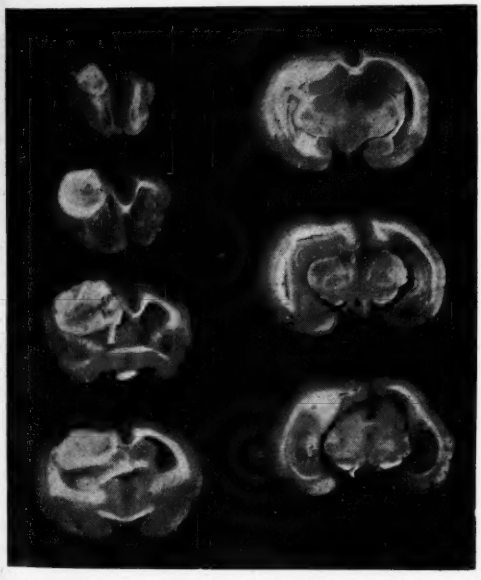

FIG. 282.—Coronal sections showing abscess 4 weeks after inoculation with staphylococci. Pyocephalus of hind part of left lateral ventricle shown in right-hand sections.

FIG. 281.—Abscess 4 weeks after inoculation with fusiform bacilli, showing dense complete capsule. (Weigert's iron hæmatoxylin and van Gieson.) (× 11.)

Obviously the number of animals in each group is too small for a more detailed analysis, particularly as individual factors in the different animals led to occasional effects that were not in harmony with the remainder. A good example of this was seen in a month-old staphylococcal abscess of considerable size (*Fig.* 282) which ended the life of the animal by rupturing into the ventricle. Histologically the stage of encapsulation reached in this specimen was greatly behind that usually found in the 3-week specimens of its group.

Other Organisms Tested.—In an early stage of our investigations 2 animals were inoculated with *Bact. friedländeri* and 2 with a coagulase-negative strain of *Staph. albus*. The former organism was tried on account of the success obtained with a similar organism (designated *B. mucosus capsulatus*) by Weed et al. (1920)

in inducing leptomeningitis in cats. A fulminating meningo-encephalitis was obtained in 1 animal, the other being uninfected, The animals inoculated with the *Staph. albus* showed no reaction.

BLOOD-CULTURES

The heart-blood was cultured from 80 animals at autopsy. In 15 the organism inoculated into the brain was recovered in pure culture; cultures from the remainder were sterile (one was contaminated with *B. coli*). Positive results were obtained from 3 of 8 rabbits dying within 24 hours, from 1 rabbit which was killed at 24 hours, and from 9 of 11 rabbits dying between 24 and 48 hours after inoculation. Cultures from animals examined within 48 hours inoculation were positive in 7 of 7 cases of pneumococcal infection, 5 of 9 cases of streptococcal infection, and 1 of 8 cases of staphylococcal infection. In addition one positive culture of *Str. pyogenes* was obtained at 5 days in an animal receiving sulphanilamide and inn which symptoms of meningo-encephalitis became manifest on the fourth day. A positive culture of *Staph. aureus* was obtained at 8 days in an animal in which a cerebral abscess was complicated by extensive suppuration in the subdural space. It is noteworthy that this animal appeared to be improving clinically at the time that it was killed. This and another example, infected with Type XIX pneumococcus, which broke its back in a fit and was therefore killed, show that the invasion of the blood-stream by organisms was not necessarily of an agonal character. Examination of the abdominal organs in 8 of these cases showed a variable degree of acute inflammation of the pulp of the spleen, but the changes in the liver and kidneys were confined to grades of parenchymatous degeneration and, in the liver, occasional focal necroses. Cocci were sometimes numerous in the capillaries in both organs, but were unaccompanied by cellular inflammatory reactions.

Of 30 cultures taken on the ninth day or later none was positive. Thus, on analysis, bacteriæmia occurred very often with pneumococcal, fairly frequently with streptococcal, and rarely with staphylococcal infections. It was invariably associated histologically with a diffuse pyogenic inflammation within the cranium, i.e., meningitis or meningo-encephalitis.

CULTURES FROM ABSCESSES

During the earlier experiments in which streptococci were used the rabbits which were killed were perfused with formaldehyde before the skull was opened in order to provide optimal material for histological study. Streptococci were, however, cultured from cerebral abscesses in three of these rabbits. One of these was killed on the fourth day after inoculation, one died on the eighth day, and the third was killed on the twenty-first day. An unsuccessful attempt was made to culture streptococci from a 6-months' resolving abscess in an unperfused brain. No cocci were seen in Gram-stained sections of this, nor of the other 6-months' resolving abscess. Gram-positive cocci were, however, seen in the sections of 5 abscesses studied in the first month, of 3 abscesses in the second month, and 1 abscess in the third month. It is therefore certain that streptococci survived in one abscess for three weeks and probable that they survived in other cases for periods up to three months.

With other organisms some of the rabbits killed were not perfused and cultures were made from their abscesses in order to find whether viable organisms were present. In this way *Staph. aureus* was cultured from 5 abscesses in the first month, and 1 abscess in the third month; fusiform bacilli from 1 abscess in the first week, 1 in the fourth week, and 1 in the second month; and anaerobic streptococci from 2 abscesses in the third week and 2 in the second month. These results, together with the demonstration of organisms in sections, showed that viable organisms persisted in the abscesses for considerable periods, and that the pathological findings in the brain represent stages in the evolution of an infective process and not merely the reaction to a transitory infection.

REVIEW OF PATHOLOGICAL EXAMINATION

In our experiments infection of the brain occurred in 75 per cent of animals. Abscesses were obtained in the majority (64 per cent) of these, while diffuse inflammatory reactions were found in the remainder (*Table II*). An established infection of the brain substance usually progressed within 48 hours in one of two directions: either the inflammatory reaction spread until the ventricle was reached or it became circumscribed. The spreading infection was accompanied by considerable swelling of the brain, which formed a hernia at the site of the decompression, and was characterized histologically by the extension of irregular areas of necrosis, often without a limiting zone of leucocytic reaction, from the site of inoculation into the white matter. In places the appearances suggested that leucocytes had formed a temporary barrier against this spread, but had become swamped by extension of the necrosis. Large numbers of organisms occupied the necrotic tissue and tended to encroach beyond its limits into the surrounding living brain.

As already noted (p. 247) herniation of the brain in rabbits is associated with subcortical changes (hæmorrhage and necrosis) which lead eventually to cyst formation in uninfected animals. The amount of necrosis observed in the white matter may thus have been in excess of that which might be expected in the absence of a bony decompression. But the production of a spreading meningo-encephalitis is not directly

attributable to this factor, as shown by its occurrence in one of the 6 animals inoculated with staphylococci through a small burr hole.

Positive cultures were often obtained from the heart-blood at autopsy in animals suffering from meningo-encephalitis or meningitis alone.

The more circumscribed inflammations which were destined to become abscesses were initially of a similar character, but acquired, within the same period of time, an orderly relationship between the necrotic process and the leucocytic reaction. In the first phase (up to 48 hours) the polymorphonuclear leucocytes were principally engaged; they became mobilized to form the central purulent area of the abscess and also collected outside the bordering zone of necrosis which represented the tendency of the abscess to spread centrifugally. After 48 hours the defence appeared to pass over progressively to the microglial cells, the amœboid forms of which are clearly capable of ingesting bacteria as well as other particulate matter. The migration of these cells centripetally into the peripheral zone of necrosis ultimately obliterated the latter, bringing the growth of the abscess to a standstill. Organisms were confined to the central purulent area; in no instance were they found peripheral to the zone of necrosis and here they were usually within microglial cells. In the meantime the vascular reactions in the periphery resulted in the provision of a fibrillar capsule, first of reticulum fibres and later of collagen, together with an inflammatory exudate composed largely of lymphocytes and plasma cells. It is noteworthy that these reactions did not proceed simultaneously on all aspects of the abscess. The process of encapsulation always began in the cortex (*Fig.* 270), especially near the leptomeninges, and could be detected here towards the end of the first week. It took place last in the white matter, being tardiest near the ventricle. Thus the zone of necrosis, representing always a potential route of spread, sometimes persisted at this deepest point up to the end of the third or fourth week after inoculation even when the remainder of the abscess had acquired a substantial wall (*Figs.* 276–279). This accounted for the late development of pyocephalus and purulent meningitis in otherwise stabilized abscesses, such as occurred in a staphylococcal example at one month (*Fig.* 282). The slower development of a capsule in the white matter is explained by the relative paucity both of microglial cells and of blood-vessels in this part of the brain. For the same reason loculation of an abscess, though rare in our material, appeared to represent the breaking out of infection into an adjacent part of the white matter as an alternative to rupture into the ventricle.

From the time that encapsulation is complete the process of resolution may be expected to set in gradually. This took place in our material through the progressive centripetal migration of macrophages, probably of microglial origin, to form foam-cells. These cells were accompanied by capillaries, fibroblasts, and lymphocytes. The clearing away of the pus and its replacement by scar tissue completed the final stage.

This process of abscess-formation varied remarkably little with different types of infecting organism. In particular we found no retardation in the encapsulation of abscesses due to Gram-negative and anaerobic bacteria.

It might be argued that the use of agar in our inoculum was responsible for the apparent uniformity of our results and it is therefore necessary to examine this possibility.

Agar Controls.—In 5 rabbits an injection of 0·1 c.c. of agar was made into the cerebrum through a burr-hole. These foci were examined histologically at the end of 2 days in one and after 5 weeks in the remainder. It was observed that a large part of the inoculum flowed back along the needle-track into the leptomeninges. In 3, however, a circumscribed mass remained in the depths of the cortex, while in the remaining 2 a shallow wedge of superficial cortex was occupied by the agar in continuity with the residue in the leptomeninges. In addition, residual agar was identified at the site of inoculation in 5 of our negative experiments with organisms. These foci were examined histologically at times varying from 8 days to 3 weeks after the inoculation. In a surprisingly large number of our negative experiments twe were unable to find any trace of the inoculum hough in some we found a linear pigmented scar, corresponding to the track of the needle.

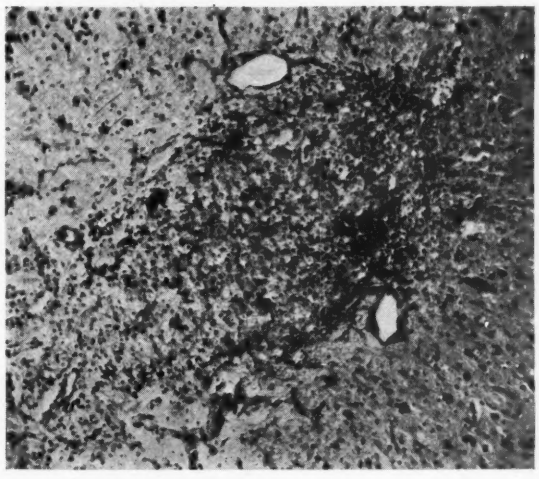

FIG. 283.—Residual lesion composed of foam-cells 3 weeks after intracortical injection of agar. (Hæmatoxylin and eosin.) (× 68.)

In the early stages (2 days) the mass of agar within the brain was surrounded by a zone of recent hæmorrhage, but there was no purulent reaction. Early stages of microglial activity were observed in the neighbouring cortical tissue. Later on the agar was gradually replaced by foam-cells of microglial origin (*Fig.* 283). Associated with this was a variable number of foreign-body giant-cells such as were also observed in the walls of the abscesses at the later periods. The replacement of the agar took place slowly, and in some specimens was incomplete even

at the end of 5 weeks. In no instance was there any encapsulation of the area, such as occurred in the abscesses. At most there was an uneven and disorderly arrangement of scanty collagen and reticulum fibres amongst the foam-cells forming the periphery of the focus, and outside this there was a variable amount of gliosis and slight proliferation of blood-vessels.

It appears, therefore, that the specific effect of agar alone upon the brain tissue is the mobilization of microglial cells which ultimately become

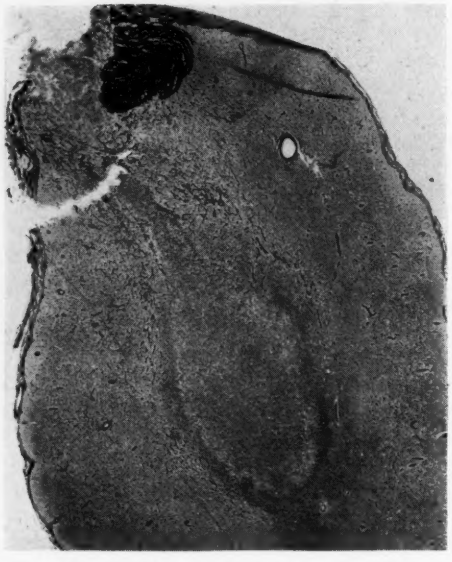

FIG. 284.—Abscess 8 days after inoculation with staphylococci, showing early development of reticulum fibres about dorsal, intracortical part of abscess. (Silver carbonate for reticulum.) (× 12.)

compound granular corpuscles, and the formation of foreign-body giant-cells, apparently from the same source. There is no excitation of polymorphonuclear leucocytes, nor is there any appreciable production of collagen and reticulum fibres. There is thus no evidence that the encapsulation of the abscesses in our experiments can be directly attributed to the use of agar.

EFFECTS OF INTRODUCING THOROTRAST (COLLOIDAL THORIUM DIOXIDE) INTO CEREBRAL ABSCESSES

Thorotrast is sometimes injected into the cavity of cerebral abscesses in human subjects in order to observe radiographically subsequent changes in the contours of the lesion and thus obtain a guide to treatment (Kahn, 1939). We have observed that such abscesses, when finally enucleated, usually possess a thick capsule in which large macrophages, stuffed with granules of thorotrast, are a predominant feature. The impression had been gained, from this clinical material, that the histological picture might be modified by the use of thorotrast, and we therefore explored this possibility in a series of experimental abscesses.

In all instances the organism used was *Staph. aureus*, because experience had shown that this was most likely to yield a suitable focal infection. The inoculum was prepared as before, except that a proportion of thorotrast, varying from a half to about one-tenth of the total volume, was mixed as intimately as possible with the agar immediately before the injection was made. In each batch of 5 or 6 animals inoculated 2 or 3 received cocci and agar alone and were kept as controls.

FIG. 285.—Cf. *Fig.* 284. Abscess 8 days after inoculation with staphylococci and thorotrast, showing reticulum fibres at ventral border of abscess as well as in cortex. (Silver carbonate for reticulum.) (× 12.)

Altogether 14 animals received thorotrast, and of these 3 were not infected, 8 developed abscesses, and 3 showed diffuse inflammatory reactions (1 meningo-encephalitis, 1 meningitis, and 1 subdural abscess). Of the 10 controls 3 were not infected, 6 developed abscesses, and 1 showed meningo-encephalitis. The animals with abscesses were killed after either 8 days or 3 weeks, with the exception of one control which died after 3 days.

Early stage (8 *days*).—There were 5 thorotrast abscesses in this group, and the 3 controls were supplemented by 3 additional specimens from our main staphylococci series, including one at 7 days and one at 9 days.

Microscopical examination showed that while a good deal of the thorotrast was retained in the agar at the centre of the abscess, a large proportion of the granules had gravitated to the ventral border of the cavity. Granules were sparse in other parts of the wall. Comparison with the controls showed that this distribution of the thorotrast granules along the ventral border was associated with a precocious development of delicate reticulum fibres often involving a segment in which the abscess lay in relation to white

matter (*Figs.* 284, 285). Associated with these fibres was an unusually abundant infiltration with microglial cells, many of which had ingested granules of thorotrast. In places the granules had migrated centrifugally for distances up to 1 to 2 mm. beyond the confines of the central purulent area, but they were nowhere accompanied by cocci, which, as in the controls, remained in the central pus or in the necrotic zone bordering it. The early stages of capsule-formation about the dorsal, intracortical part of the abscess corresponded quite closely to those seen in the controls. In one specimen alone the precocious mesoblastic reaction was lacking at the ventral pole of the abscess: in this instance very little thorotrast was present, the greater part having escaped into the ventricular system at an earlier stage.

Late stage (3 *weeks*).—There were 3 thorotrast abscesses in this group and 2 controls which were supplemented by a third from our main staphylococcal series.

There was no obvious difference at this stage between the thorotrast abscesses and the controls. In both groups there was one example in which a segment of the wall, towards the ventricle, showed no encapsulation. The example containing thorotrast was of unusually large size and had ruptured into the ventricle, with the production of a focal pyocephalus. This animal developed a right hemiplegia and appeared almost moribund on the third day after inoculation, but recovered and appeared in good health when killed. Microscopically the greater part of the thorotrast appeared to have entered the ventricle, where it was loculated by the choroid plexus. There was a conspicuous excess of reticulum fibres in the stroma of the plexus and the posterior part of the ventricle was greatly expanded.

Comment.—The introduction of a small quantity of thorotrast with the inoculum had the effect of stimulating early stages of capsule-formation about the ventral border of the abscess, while in control specimens such stages were confined to the dorsal border. This precocious development appeared to be associated with the gravitation of the thorotrast granules to the dependent part of the cavity; here they penetrated the adjacent nervous tissues and provoked the mobilization of microglial cells by which they were ingested. The quantity of thorotrast injected in our experiments was much smaller proportionately than that used in human cases; hence it is perhaps not surprising that the appearances of the 3-week thorotrast abscesses were not obviously different from those of the controls.

It was noteworthy that the granules of thorotrast which penetrated the tissues were not accompanied by cocci. Comparison with the controls showed that the numbers and distribution of the cocci in the thorotrast abscesses were similar. There was no evidence that thorotrast *in vivo* exercised a bactericidal effect. This was confirmed by *in vitro* tests. Thus thorotrast (2 per cent) did not diminish the number of colonies of staphylococci or hæmolytic streptococci developing in poured plates. In broth or serum broth 2 per cent thorotrast did not inhibit the growth of staphylococci, hæmolytic streptococci, anaerobic streptococci, or fusiform bacilli. After incubation of the broth tubes the thorotrast was found to have settled as a flocculent deposit. Films of this deposit showed very numerous organisms among the thorotrast granules.

SULPHANILAMIDE THERAPY OF STREPTOCOCCAL ABSCESS

An attempt was made to discover whether the parenteral administration of sulphanilamide would influence the character of the lesions produced by the strain of streptococcus which we were using. Twenty-six rabbits were inoculated in the usual way with *Str. pyogenes*; 13 were treated with sulphanilamide and 13 reserved as controls.

The streptococcus used was shown to be sensitive to sulphanilamide by the ditch-plate method (Fleming, 1940–1). Sulphanilamide (Streptocide: Evans, Lescher & Webb) was injected intraperitoneally in a dose of 300 mg. per kilo body-weight (Hudson, Smith, and Selbie, 1941). We injected the first dose three hours after the inoculation of the streptococci and repeated it on the two succeeding days. Two rabbits received further doses on the fourth and fifth days after inoculation. Satisfactory concentrations of sulphanilamide were found in the blood three hours after the injection of the drug, but we obtained some evidence that the blood concentration of the drug fell below the inhibitory level in the latter part of the 24-hour period between doses. The results of the experiments are given in *Table III*.

It is possible that sulphanilamide might be shown to influence experimental infections if a

Table III.—Results of Inoculations of Hæmolytic Streptococci, with and without Sulphanilamide Therapy

	No. of Experiments	No Infection	Meningo-encephalitis	Meningitis	Meningeal Abscess	Cerebral Abscess	Deaths	
							No.	Day of Death
Treated	13	3	3	1	3	3	4	1, 4, 5, 11
Controls	13	2	3	2	1	5	3	1, 2, 2

more virulent strain were inoculated into a larger number of animals and an inhibitory concentration of the drug maintained in the blood for several days after inoculation. Under the conditions of our experiments no significant clinical or pathological differences were observed between the treated and control animals.

DISCUSSION

Attempts to produce experimental brain abscesses have previously been made by Homén (1913), Essick (1919), Groff (1934), and others, but with the notable exception of Homén's work, these studies have all been confined to early stages of abscess formation. Homén obtained abscesses in dogs, rabbits, and guinea-pigs by the direct inoculation of the brain with a mixture of a streptococcus, staphylococcus, and *B. perfringens* (*Cl. Welchii*). His results in a series of 30 dogs are given principal place in his report. Stages in abscess formation were observed at periods varying from 5 hours to 2 months, but in animals which lived longer he found residual foci without suppuration. Capsule formation began at 5 to 7 days and there was no evidence of progressive inflammation after that time. Regressive changes in the inflammatory process were apparent after 17 days and it was then no longer possible to demonstrate organisms in microscopical preparations. Culture of the abscesses in the first four days yielded all three organisms, but after 6 days cocci only were obtained and after 25 days the abscesses were sterile. Homén's series of rabbits, of which details are not given, seems to have provided stages of abscess formation from 6 hours to 6 months; regressive changes apparently made a tardier appearance than in the dog. Although *Cl. Welchii* did not survive in the lesions longer than in the dog, it was possible to culture cocci up to 6 months. Homén gave no details of the reaction around the abscesses in the rabbit, but stated that no proper encapsulation took place: all components of the capsule were more feebly formed and appeared later than in the dog, which, according to Homén, approximates more closely to the human subject in this respect. The cytological features of the canine abscesses are described by Homén with exquisite precision, stage by stage.

His work, however, antedated that of the Spanish school on the glia and the silver impregnations for reticulum fibres. In the present study these more recent techniques have been used to supplement the routine stains with aniline dyes. By means of them it has been possible to observe the earliest stages of encapsulation and the specific part played by the microglia in the phagocytosis both of bacteria and of the products of tissue breakdown. The phases in the evolution of our experimental abscesses have been reviewed above (p. 249). Although this work has been confined to the rabbit, the similarity of the histological appearances to those observed in different stages of abscess formation in man permits the discussion here of certain clinical applications. One of the salient features of our experimental abscesses was the mode of spread by means of a peripheral zone of necrosis to which the infecting organisms might gain entrance by centrifugal spread but were thereupon phagocytosed by centripetally migrating microglial cells. Such a tendency to spread became limited by the deposition of reticulum fibres in the tissues peripheral to the zone of necrosis, and this formation took place first along the intracortical segments of the abscess, especially near the leptomeninges. It took place last in the deepest segment in the white matter, that is, towards the ventricle. The clinical significance of this is clear: enucleation of an abscess as a routine method of treatment (Vincent et al., 1937) depends for its success upon the existence of a sufficiently resistant wall at this deepest point, and the risk of rupture of the abscess during enucleation persists until the wall is complete. The formation of such a wall is predominantly a matter of time, but, as shown above, individual variations prohibit any dogmatic prognosis as to the stage of encapsulation to be expected at any given period of the infection. Clinical experience, however, suggests that a firm capsule should be found at the end of three months (Vincent et al., 1937), and we can confirm this in both a streptococcal and staphylococcal specimen examined at this period. But it should be remembered that the existence of a tough capsule in the superficial part of the abscess is no guarantee of a similar formation in the deepest segments.

The slower progression of encapsulation in the white matter accounts for two features of cerebral abscesses in man—namely, their tendency both to become loculated and to extend towards the ventricle. Loculation results from a spread of infection from the primary focus before encapsulation is complete. It was observed in only 3 examples in our series. It is more frequent in human abscesses, probably on account of the relatively greater depth of the white matter and its subdivision by sulci. Examination of such human specimens usually reveals a diminishing density of the wall and greater fluidity of the pus in passing from the more superficial to the deeper loculi. The most superficial focus may, indeed, be dried up while the deepest is still indifferently encapsulated. Such a progression of the infective process into the depths of the white matter had clearly taken place in the patient whose lesion, a staphylococcal abscess of 6 months' duration, is diagramatically represented in *Fig.* 286. Evidence of spread towards the ventricle was found in many of our early specimens and also in a month-old staphylococcal abscess which terminated by intraventricular rupture.

Vincent et al. (1937) have advocated a wide bony decompression in the early treatment of

cerebral abscesses, suggesting that the consequent relief of increased intracranial pressure helps in the localization of the infection. In all except 6 of our rabbits the presence of a relatively large decompression may have permitted the survival

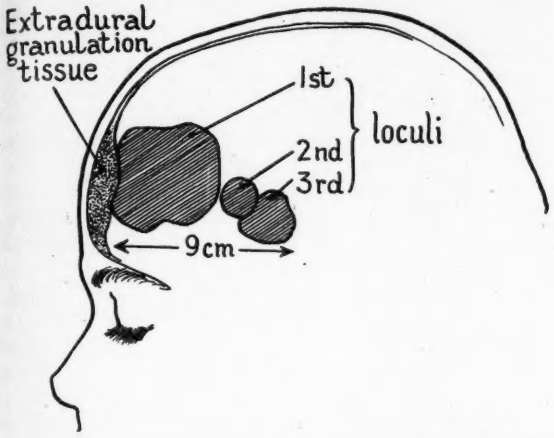

FIG. 286.—Female, aged 16. Diagram of trilocular left frontal abscess (culture: *Staph. aureus*) removed at operation 6 months after initial infection from a stye. The deepest abscess had a thin wall. (*Professor Caivn's case.*)

of a certain number of animals which would otherwise have died in the acute stage. But in the remaining 6 animals we inoculated the brain with *Staph. aureus* through a small burr hole, and in 3 of these a localized infection was obtained (1 meningeal and 2 cerebral abscesses). The two cerebral abscesses were examined histologically a week later and showed the usual early stages of encapsulation. We have obtained no evidence, therefore, that localization of infection and its subsequent encapsulation are directly dependent upon the coexistence of a large decompression. But a decompression allows the œdematous cerebral tissues to expand without undue compression of the rest of the brain. In rabbits, at any rate, bulging at the site of decompression denoted œdema, and was proportionate to the severity of the inflammatory reaction. When the acute stage had subsided the decompression became flat and remained so even though an abscess of considerable size might have formed in the subjacent brain. Similarly with organisms of relatively low pathogenicity, such as the anaerobic and Gram-negative bacteria, a large abscess might form without palpable swelling of the brain. Similar observations are made from time to time in human cases of cerebral abscess: in the absence of œdema of the adjacent brain tissue there may be little increase in the total volume of the brain. In this department the tendency within recent years has been to depend upon aspiration in the preliminary treatment of brain abscess, and to perform decompression in those cases only where aspiration is insufficient to relieve increased intracranial pressure (Pennybacker, 1941, short communication).

Our experiments with thorotrast indicate that this substance may be helpful not only for demonstrating an abscess radiographically, but also in promoting capsule-formation where it is most needed. Thus the particles gravitate to the dependent part of the abscess cavity and, by penetrating the tissues for a millimetre or two, stimulate the microglial cells and the production of reticulum fibres. It might therefore be desirable, in human cases of cerebral abscess, to orientate the head of the patient in such a way that gravitation occurs towards the ventricle, though the risk of leakage of the thorotrast into the ventricular system, with a resulting aseptic meningitis, should be carefully weighed.

Our experiments have provided no evidence that abscesses due to anaerobic and Gram-negative bacteria are less well encapsulated than those due to Gram-positive cocci. On the contrary, capsule-formation appeared to be relatively retarded in the presence of a highly pathogenic organism such as the hæmolytic streptococcus as compared with organisms less pathogenic to the rabbit's brain such as *P. vulgaris* and the fusiform bacillus. These observations do not conform to current belief. But we have found no reason to suppose that the conditions of our experiments, and in particular the use of agar in our inoculum, have been responsible for these results. Nor do we consider that they are peculiar to the rabbit. Thus from recent observations in human material we can testify to the presence of a well-formed collagenous capsule in abscesses due to fusiform bacilli and anaerobic streptococci (1 case), *P. vulgaris* combined with Gram-positive cocci (1 case), and *P. vulgaris* and anaerobic streptococci (1 case); in none of these abscesses was thorotrast used for radiographic demonstration.

SUMMARY

1. Abscesses have been experimentally induced in the brain in 63 out of 130 rabbits inoculated with a variety of organisms pathogenic to man. Diffuse inflammatory reactions occurred in 35 others.

2. Histological stages in the evolution and resolution of cerebral abscesses have been traced up to 6 months after inoculation.

3. Encapsulation began first towards the surface of the brain and was completed last towards the ventricle.

4. No deficiency in encapsulation was found with Gram-negative and anaerobic organisms.

5. The introduction of thorotrast with the inoculum had the effect of stimulating capsule-formation at the dependent part of the abscess cavity.

The expenses of this work were defrayed by a grant from the Medical Research Council, to whom our thanks are offered. We also thank Professors J. A. Gunn and H. W. Florey for

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CRITICAL REVIEW

SLOW UNION OF FRACTURES

WITH A STUDY OF 804 FRACTURES OF THE SHAFTS OF THE TIBIA AND FEMUR

BY R. WATSON-JONES
CONSULTANT IN ORTHOPÆDIC SURGERY, ROYAL AIR FORCE
AND WING-COMMANDER W. D. COLTART
AUXILIARY AIR FORCE

THE test of success in the treatment of a fracture is the quality of end-result rather than the time taken in achieving the end-result.[1] It is better that a fracture should unite soundly and without disability in four months, than that it should unite imperfectly and with deformity in two months. Time is not saved when recovery is accelerated at the cost of permanent disability; a disabled man loses time every day of his life. But although prevention of permanent disability must be the first consideration, prevention of unduly prolonged disability must be the second. The disability period is important in the industrial demands of peace and still more important in the industrial and military demands of war.

Of the many factors which influence the disability period the most constant is the time taken for the fracture to unite. As a broad generalization it may be estimated that the duration of total disability is half as long again as the time taken for union of the fracture. If a fracture of the tibia unites in ten weeks, recovery is usually complete after a further period of treatment of about five weeks; whereas if ten months elapse before union is sound, nearly five months is needed before normal joint movement, strong muscle power, and full confidence in the limb are restored. It is doubly important, therefore, to prevent unnecessary delay in the union of a fracture, and doubly important to investigate the recent challenge that modern methods of fracture treatment are responsible for serious delay.

The impression that fractures of the shafts of the femur and tibia are uniting more slowly than in former years has gained wide acceptance. At a meeting of the British Orthopædic Association[2] "most speakers remarked on the impression generally held that delay in union is more common than formerly". McMurray's[3] "impression that the rate of union of fractures of the tibia is much slower than previously, has become a conviction". Bonnin[4] asks "What has caused the delay from consolidation in six weeks, to consolidation in eleven weeks, between 1840 and 1940?" Page[5] believes that "delayed union in fractures of the tibia and femur has been more in evidence for the past ten years than it was in the previous twenty". Burns and Young[6] think that "deterioration in the rate of union came about not recently nor gradually, but suddenly in 1930". A *Lancet* annotation[7] warns us "that recent advances may have been in the wrong direction".

The surgical literature of the last thirty years appears to give undoubted support to this impression. Every text-book on fractures published before 1910 records the time required for union of fractures of the tibia as less than eight weeks. Heister[8] estimated the period at thirty to fifty days, Malgaigne[9] at thirty-five to forty days, and Hamilton[10] at thirty days in

ordinary cases, and forty days "when union was delayed". On the other hand, Böhler,[11,12] writing in 1935, finds that "fractures of the tibia with displacement take ten to twelve weeks" and that "fractures between the middle and lower thirds often require fifteen to twenty weeks". The fractures investigated for this review showed an average time of union of all fractures of the tibial shaft of nineteen weeks. In seven unpublished series of which we have information, totalling 1074 fractures of the tibia in patients aged 15 to 45 years treated during the last twelve years by six different surgeons, the average time of union was 13 weeks, 16½ weeks, 15 weeks, 17 weeks, 25 weeks, 22 weeks, and 25 weeks respectively. Is it then true that methods of treatment introduced in recent years have doubled or even trebled the time of union of fractures of the tibia? McMurray[3] is convinced, and he blames the unpadded plaster cast because its pressure "prevents the formation of the hæmatoma which should be present in every fracture". Hobson[13] is convinced, and he blames the practice of complete immobilization. Other surgeons have also shown lack of faith in immobilization: "I think the case for slight movement at the fracture site hastening union can be defended"[4]; "absolute immobilization is not essential to bony union"[5]; "the difference between splints and plaster is that immobilization is much less complete in splints; is it not possible that the quicker union of those days was due to this very lack of early fixation?"[6] Must we therefore dismiss so soon the very recently accepted principle of complete and uninterrupted immobilization in the treatment of fractures, a principle based on the teachings of a long line of menders of the maimed in British medicine—Hilton, Hunter, Hugh Owen Thomas, and Robert Jones? Must we dismiss it despite the fact that slow union is suspected only in fractures of the tibia and femur, that fractures in the upper limb which are also treated by complete immobilization in plaster are uniting as quickly as they did, and that fractures of the femur are seldom treated in plaster at all? Surely if there is truth in the impression that fractures of the shafts of the femur and tibia are uniting more slowly, we must seek for a cause which is common to the treatment of these two particular fractures. But before accepting the charge that recent methods of treatment have doubled or trebled the time of union, two great changes in the principles of fracture treatment must be examined, changes which distinguish the practice of to-day from that of thiry years ago: (1) the present tendency to demand evidence of radiographic consolidation rather than clinical union alone; (2) the present belief that all fractures unite if they are immobilized long enough, and that non-union is never inevitable.

1. Clinical Union and Radiographic Consolidation.—In former years it was customary to remove splints at the earliest possible moment, usually at the first sign of clinical union; to-day, protection in plaster is continued until the patient is able to walk, run, and play games, and often until there is radiographic evidence of consolidation. 'Clinical union' as shown by disappearance of local tenderness and freedom from elasticity or springing of the fragments[14] is often reached within six or eight weeks of injury; 'radiographic consolidation' as proved by uniform calcification of the callus is not completed until many weeks later. This is true to-day and it was true in former years. We must not compare the time required for early clinical union thirty years ago with the time required for radiographic consolidation to-day. Fractures of the tibia were never consolidated in forty days; they were never soundly and safely united in six or eight weeks; they were united only firmly enough for splints to be removed and for the patient to be discharged from hospital, after which time the fracture went on to firm union, malunion, or non-union according to the stage of repair which had been reached in that short period.[15] This is proved by the literature of the very day when union was supposed to be occurring with such rapidity. In 1912 Robert Jones[16] wrote: "We must realise that the academic period of consolidation authoritatively asserted is not accurate, and that bones which appear firm to the hand will yield after many weeks to the incidence of body-weight". Again, he wrote: "Malunion and non-union are common occurrences. I see such cases every week. We are misled by our text-books as to the period of complete consolidation of bone". An editorial leader in the *British Medical Journal*[17] of thirty years ago reads: "The time for consolidation of a long bone after fracture should be revised, that is lengthened, since yielding of the fracture often appears after the case has passed out of the hands of the surgeon". The lesson has now been learned. Protection of recently formed callus is no longer discontinued after six or eight weeks, but only after ten or twelve weeks, not because fractures are uniting more slowly, but because immobilization is deliberately continued until a later stage of union. In consequence of this more lengthy protection, the complications of malunion and non-union which were so common thirty years ago have virtually disappeared. Moreover, final recovery has been accelerated. There can be little doubt that patients whose plasters are retained a few weeks longer than is strictly necessary for union, and who are taught to walk, run, and play football or cricket before the plaster is removed, regain confidence and overcome disuse changes more rapidly than patients who lose their protection at an earlier date. The conclusion is irresistible that when a fracture of the tibia is treated by simple manipulation and plaster and it is recorded that union took place in ten or twelve weeks,

the fracture has united as quickly as ever it did in the past, and that neither the unpadded plaster cast nor the practice of uninterrupted immobilization has been responsible for delay.

2. Non-union is not Inevitable.—The second recent advance in fracture treatment which has led to confusion is the recognition that non-union is never inevitable,[18] and that every fracture will unite if it is immobilized long enough.* In former years a certain number of weeks was fixed as the time required for union of a fracture, and splints were removed at that date regardless of the stage or repair which had been reached; if the fracture failed to unite, non-union was believed to have been inevitable. It is now recognized that the rate of union of a fracture is influenced by so many factors, and that so many combinations of delaying influences are possible, that no fixed time for union can be laid down. Only the minimal period can be fixed. After immobilization for the minimal period a certain proportion of fractures are soundly united; if immobilization is continued a month or two longer many more are united; if immobilization is continued long enough every fracture unites. Sometimes, however, particularly when there is infection of the fracture, immobilization must be continued for six or twelve months. In fractures with complete loss of blood-supply to one fragment (such as fractures of the neck of the femur) it is now known that union may be sound only after two or more years of continuous immobilization. If all fractures are grouped together and an average time of union is calculated, the figure is hopelessly misleading. It is entirely without significance to say that in 800 fractures investigated for this review, the average union time of fractures of the tibia was 19 weeks. It would be as useful to record that the average age of the population of this country is thirty, or forty, or whatever it may be. It means nothing to find that 1074 fractures of the tibia had an average time of union of 17 weeks. The figure depends wholly upon the proportion of simple uncomplicated fractures uniting in about ten weeks, difficult fractures uniting in ten to twenty weeks, infected fractures uniting in six to twelve months, and avascular fractures uniting in one to three years. It is meaningless to compare these figures with the figures of thirty years ago which averaged only the more rapidly uniting fractures. The principle of uninterrupted and prolonged immobilization, far from being assailed by such comparison, is simply shown to have been responsible for almost complete disappearance of the non-union which was formerly accepted. Of the cases under review which were treated in Royal Air Force hospitals, despite a high proportion of multiple, gravely comminuted, seriously contaminated, and heavily infected fractures, there was not one case of non-union. To the surgeons of thirty years ago this would have seemed incredible; to the surgeons of to-day who are unfamiliar with the practice of prolonged and uninterrupted immobilization of infected as well as closed fractures it may still seem incredible; nevertheless it is true.* Occasionally early bone-grafting was performed with the object of accelerating union when it was surmised that the fracture would be of the slowly uniting type; but in a consecutive series of fractures treated by many different surgeons without such acceleration there was not a single case of established non-union. Similar results have been reported by Soulié and Linares,[19] Trueta,[20] and by d'Harcourt,[21] who stated of 5000 fractures treated at Vallcarca, "Nous n'avons observé ni pseudarthrosis ni difformités". Jolly[22] also reported on Spanish war casualties and observed that "absolute immobility, attainable by plaster and by no other means, has almost banished the bugbear of pseudarthrosis so often seen in the war of 1914–18".

The principle of complete and continuous immobilization of shaft fractures is therefore vindicated. Fractures of the rapidly uniting type, although they are now treated by immobilization in plaster, are uniting as quickly as in former years; fractures of the slowly uniting type, because they are now treated by long-continued immobilization in plaster, are uniting with greater certainty than in former years. But that is not enough. Is it possible that the proportion of slowly uniting fractures is greater than it was? If there has been an increase in the frequency of slow union, is it inevitable, or is it a consequence of modern methods of treatment? What are the causes of slow union?

CAUSES OF SLOW UNION OF FRACTURES

General Causes.—A vast literature has accumulated on the subject of constitutional and dietetic errors in relation to the union of fractures. Consideration has been given to calcium and phosphorus metabolism,[23–26] diet and general metabolic deficiencies,[27] parathyroid and ovarian secretions,[28–31] local and plasma

* We must of course recognize the exception of many pathological fractures and other fractures in which there is no ossifiable medium between the fragments—for example, fractures with interposition of living soft tissue between the bone ends, and fractures in which early removal of the whole thickness of the shaft of the bone causes collapse of the periosteal tube with obliteration of the fracture-hæmatoma. If in these fractures an ossifiable medium is restored between the fragments, it is then true to say that every fracture will unite if it is immobilized long enough.

* A few cases were invalided before union was complete, and we do not know the final result. It will also be seen from *Table I* that 35 fractures in the series were not united at the time of investigation; most of these are now united, but not yet all.

phosphatase,[32-34] and vitamin deficiencies.[35,36] It is understandable that in time of war attention should turn once more to the question of food and vitamin supplies,[37, 38] and that in a search for the causes of slow union, ascorbic acid estimations should be made. But slow union has been suspected only in fractures of the tibia and femur and not in fractures as a whole; the slowness was noted before the war; and despite war, the general standard of nutrition in this country, shared by rich and poor alike, is probably no less high than it has ever been. Moreover fracture-healing is essentially a local process.[39-41] The fracture-hæmatoma is a closed chemical factory, mobilizing and depositing salts in response to local tissue biochemical reactions, and almost uninfluenced by general blood-calcium and blood-phosphatase levels, the secretion of endocrines, or the supply of vitamins.

Local Causes.—In an attempt to answer the problems of slow union, an investigation has been made of 800 fractures of the shafts of the tibia and femur treated in orthopædic centres of the Royal Air Force Medical Service and the Liverpool Royal Infirmary.* The actual figures are shown in *Tables I–VIII*; they have been subjected to the tests of statistical analysis and are of proved significance. Of the many local causes of slow and delayed union, three are outstanding in their effect: (1) Interrupted immobilization; (2) Infection of the fracture; and (3) Distraction of the fragments. These influences often coincide with each other and with other delaying factors. In no fracture which has been investigated has it been possible to say that slow union was due to one cause and one alone. A fracture which is infected may also be distracted; both infected and distracted fractures are often subjected to interrupted immobilization. The calculation of an 'average union time' is therefore no less misleading in a selected group of fractures than in the whole series. Nevertheless, it is possible to estimate the gravity of each delaying factor by comparing in each group the proportion of fractures which united in 12 weeks, 12 to 16 weeks, 16 to 24 weeks, and so on. This is seen in *Table II*, which may be put in simplified form :—

TIME OF UNION OF 417 FRACTURES OF THE SHAFTS OF TIBIA AND FIBULA (*see Table II*)

		United by the			
		12th week per cent	*16th week* per cent	*24th week* per cent	*48th week* per cent
Closed :—					
Continuous immobilization	(199)	42	70	95	100
Interrupted immobilization	(104)	11	33	74	100
With distraction	(16)	—	6	31	97
Open :—					
Without infection	(46)	9	39	72	100
With infection	(52)	2	6	35	91

* It is to be noted that many fractures were transferred to these centres several weeks or even months after injury, and that errors of treatment which are analysed in succeeding pages must not necessarily be attributed to the surgeons of the Royal Air Force or the Liverpool Royal Infirmary.

It is evident that the rate of union is never constant. Although five groups have been differentiated—uncomplicated fractures, fractures with interrupted immobilization, fractures with distraction, open fractures, and infected fractures, each group is also influenced by many other factors. Even in the case of simple closed fractures which were reduced without difficulty and immobilized without interruption, union took place at very variable times from the tenth to the twenty-fourth week, these variations being due to the age and constitution of the patient, violence of injury, site of fracture, type of fracture, degree of comminution, blood-supply of the fragments, persistence of angulation, practice of too early weight-bearing, and so on. Such minor delaying factors, singly or in combination, also influence the rate of union of inadequately immobilized fractures, distracted fractures, open fractures, and infected fractures. The permutations and combinations are enormous. Who, then, will have the courage to say that the correct time to allow for the union of a fractured tibia is eight weeks, twelve weeks, or any other number of weeks? Who will perpetuate the former teaching by which "the dictates of a calendar are accepted, every fracture not united within the specified time is labelled 'ununited', and a new régime of treatment is instituted by massage, electrotherapy, hammering, damming, and calliper splints? It would be as reasonable for a gardener to uproot every plant not in flower by a certain date in order to stimulate its growth by heat and chemicals".[18] It is obviously inaccurate to suggest that simple fractures are uniformly rapid in their union and infected fractures uniformly slow. But the delaying influence of infection can be recorded accurately in the observation that whereas 70 per cent of simple fractures of the tibia are united in sixteen weeks, only 6 per cent of infected fractures are united at that time. Similarly it may be observed that if distraction occurs, no matter what other influences are concerned, no fracture of the tibia unites in three months, only a third unite in six months, and the majority unite only in six to twelve months.

Fractures of the shaft of the femur may be analysed in a similar manner by considering in separate groups, fractures with continuous immobilization, fractures with interrupted immobilization, fractures with distraction, and open and infected fractures.

TIME OF UNION OF 142 FRACTURES OF THE SHAFT OF THE FEMUR (*see Table III*)

		United by the			
		12th week per cent	*16th week* per cent	*24th week* per cent	*48th week* per cent
Closed :—					
Continuous immobilization	(75)	61	87	95	100
Interrupted immobilization	(30)*	—	10	73	100
With distraction	(14)	29	43	93	100
Open and infected	(23)	17	30	48	100

* i.e., late refracture, late rebowing, and late operative reduction.

The same delaying influences are evident. Exactly as with fractures of the shaft of the tibia, interrupted immobilization almost doubles the time of union and infection causes still greater delay. Similarly when distracted fractures of the femur are compared with uncomplicated fractures the delay is obvious. But comparison between distracted fractures of the femur and distracted fractures of the tibia shows that this complication is less harmful in the femur which is richly supplied with blood, than in the tibia which is relatively avascular. Indeed, comparison of the whole table of femoral fractures with the table of tibial fractures demonstrates in a general way that fractures of the shaft of the femur unite more rapidly than fractures of the shaft of the tibia.

CLOSED FRACTURES WITH CONTINUOUS IMMOBILIZATION

The 199 fractures of the tibia in this group were reduced promptly by manipulation and immobilized continuously without interruption. Fractures needing late remanipulation, remoulding, or late wedging of plaster were excluded. The rate of union showed considerable variation; 40 per cent were united within twelve weeks, 30 per cent between twelve and sixteen weeks, 20 per cent between sixteen and twenty weeks, and 6 per cent between twenty and twenty-four weeks. What were the reasons for this variation? Among the factors to be considered are the site of fracture in the upper, middle, or lower thirds of the shaft, the shape of fracture (spiral, oblique, or horizontal), the violence of injury and comminution of bone, the blood-supply of fragments, the accuracy of apposition and alinement, and the practice of early or late weight-bearing.

Site of Fracture.—Many more fractures occurred in the middle third than in the lower third of the bone, but the frequency of rapid and slow union was almost identical in the two groups, and it would appear that the site of fracture is unimportant (*Table V*).

Type of Fracture.—Similarly the type of fracture had little effect; the frequency of rapid union was about the same in spiral, oblique, and transverse fractures (*Table V*). Furthermore, violence of trauma and comminution of bone[40, 41] is not of major importance provided that the fracture remains a closed and not an open injury, and that no large fragment is completely deprived of blood (*Table IV*).

Impaired Blood-supply.—The effect of blood-supply is not easy to determine because there is seldom clinical or radiographic evidence by which the diagnosis of injury to the nutrient artery can be established. Nevertheless double fractures of the shaft of the tibia are characterized by slower union of the fracture remote from the nutrient artery than of the more vascular fracture near the artery,[18] and a free supply of blood must be considered no less important in tibial fractures than in fractures of the neck of the femur and the carpal scaphoid bone, in which loss of blood-supply is known to delay union. Tibial fractures with separation of a triangular butterfly fragment were usually slow in uniting; the fragment is sometimes separated from soft tissue attachments, and loss of blood-supply may be proved by the radiographic evidence of relative density which

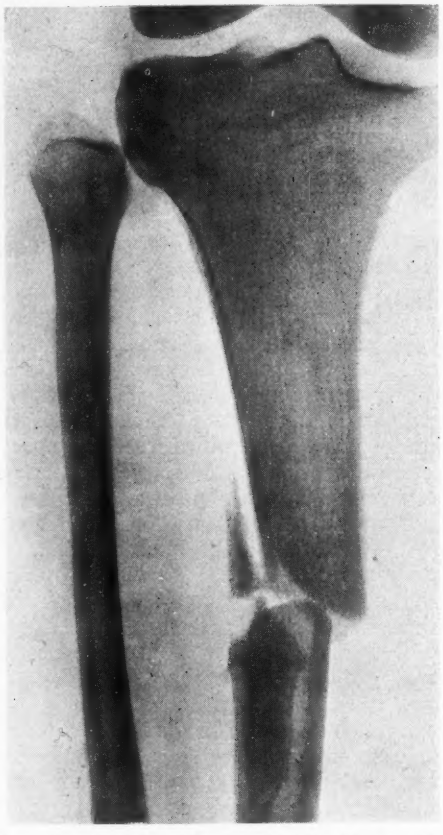

FIG. 287.—Fracture of shaft of tibia with avascular 'butterfly fragment' causing slow union.

contrasts with the decalcification of neighbouring vascular bones (*Fig.* 287). Such interposition of an avascular fragment which must undergo 'creeping substitution' before union takes place[42] is an obvious source of delay. Further evidence of the importance of blood-supply is seen in the indolence and slow union of fractures in which the fragments have been widely stripped in the course of an operative reduction, internal fixation, or a bone-grafting operation.

Angulation and Early Weight-bearing.—Fractures of the tibia with uncorrected angulation unite more slowly than fractures in perfect alinement (*Table VI*). The delay is the same whether the angulation is inwards or outwards. It is most striking when early weight-bearing is encouraged. Of 191 uncomplicated tibial fractures analysed from this point of view, over 50 per cent united within twelve weeks

when no weight-bearing was permitted before the stage of clinical union, whereas only 37 per cent united rapidly when earlier weight-bearing was allowed. The average delay was in the region of four weeks. Such delay is to be expected from theoretical considerations alone. Even if a plaster cast is unpadded, it does not prevent telescopic movement and impaction of the fragments. This was proved by F/Lt. W. R. Barrington, who inserted a metal pin in the plaster to mark a level, and noted the position of the fragments in relation to the mark in radiographs taken with and without weight-bearing. The fragments moved down within the plaster whenever weight was borne. Impaction may be harmless when the fragments are in perfect alinement; indeed, although proof is lacking, it is often believed to be beneficial; but if there is the slightest angulation, weight-bearing compresses the fragments only on the closed side of the angle and distracts them on the open side. Distraction is one of the most serious of delaying factors, and local distraction on one side of a fracture must also cause delay.* Early weight-bearing before the stage of clinical union is therefore of doubtful merit, no matter whether the fragments are immobilized in plaster or transfixed by skeletal pins incorporated in a Roger Anderson or Haines apparatus.[43–46] Functional activity, muscle development, and normal circulation can be promoted no less effectively by non-weight-bearing exercise. Early weight-bearing is to be discouraged; the gain is doubtful, the danger is real.

CLOSED FRACTURES WITH INTERRUPTED IMMOBILIZATION

The 104 fractures of the tibia and 30 fractures of the femur classified under this heading include: (*a*) Fractures in which primary reduction failed and repeated manipulation or late wedging of plaster was necessary; (*b*) Fractures in which malposition was recognized late; (*c*) Unstable fractures with late redisplacement needing secondary manipulation or operative reduction; (*d*) Fractures subjected to strain before the stage of clinical union by careless handling; (*e*) Fractures subjected to strain after the stage of clinical union, causing rebowing or refracture. If a fracture is deliberately remanipulated several weeks after injury, cellular tissue between the bone ends is torn, blood-vessels are ruptured, callus is refractured, and the process of repair must begin again. This is generally recognized, and loss of time is accepted as the inevitable cost of correcting early failures of treatment. But it is not always recognized that careless handling of a fracture at a time when it is being replastered may cause strain which amounts to remanipulation or refracture, and that this is no less certain in its delaying influence than a deliberate and necessary remanipulation. If a plaster is to be changed, there must be no interval when unguarded movements are permitted. The discarded cast must not be removed in the ward by a nurse, or in the anteroom by an assistant, but only in the theatre by the surgeon who supports the limb, protects the fragile tissues, and guards against every strain. In slowly uniting fractures and infected fractures where there is often a gap between the fragments, this is particularly important. Union can never take place if the bone is refractured at regular intervals every time the plaster is changed.

Reduction must be Immediate — not Gradual.—A frequent error of treatment which causes serious interruption of immobilization is the reduction of fractures of the shafts of long bones, and particularly of the femur, by gradual and long-continued traction. No anæsthetic is given and there is no formal manipulation or reduction of the fracture; a weight is simply suspended from the limb. Fear of overpull usually leads to the use of insufficient weight in early days; later on the weight is increased. Meanwhile the muscles have lost their elasticity and still more pull is needed. The heaviest weight is applied at the most dangerous time, two or three weeks after injury; distraction of the fragments may occur. Further adjustments are made and the fragments are remanipulated. Finally, operative reduction or a bone-grafting operation may be performed. Such a régime is based on a misconception of the principle of reduction of fractures by traction. The object of treatment should be to reduce the displacement at once and thereafter to maintain reduction without interruption. Clay Ray Murray[47,48] places great emphasis on this "timing of fracture reduction" and on securing reduction before the fracture-hæmatoma is organized and replaced by granulation tissue. He writes, "Reduction so far as length is concerned should be attained within a few hours. The author likes to get it within four hours. Every effort should certainly be made to get it within the twelve-hour period". Full relaxation of muscles should be secured by local or general anæsthesia. Over-riding must be corrected completely no matter how heavy the weight which is needed; provided that heavy weights are never left in position overnight, it is permissible to use a pull of 25, 30, or even 35 lb. Radiographic control is advisable. The fragments are manipulated into apposition, alinement is corrected, fixation is secured by local splintage, the weight is adjusted, the fragments are impacted, and before the surgeon leaves the bedside, reduction of the fracture is complete. The after-treatment then

* There is evidence to suggest that weight-bearing on an angulated bone which is avascular, and therefore indolent, is one of the important factors accounting for the persistent non-union of congenital pseudarthrosis of the tibia.

consists in maintaining reduction continuously and without interruption, splints being relied upon to prevent angulation and a weight of about 15 lb. to prevent over-riding.

Traction should Control Length but not Alinement.—Another failure of treatment recently introduced is the practice of supporting the limb on a Braun's splint, or the slings of a wide Thomas's splint, and relying on the pull of a heavy weight to maintain not only length but also alinement. It is often found that weight sufficient to prevent angulation causes distraction; if the weight is reduced to allow impaction of the fragments, angulation recurs. Repeated adjustments are made, first to correct angulation, then to overcome distraction, then to correct recurrent angulation, and so on. Union is seriously delayed. If it was not that the femur is a vascular bone, that callus formation is vigorous, and that it is difficult to stop these fractures from uniting, the dangers of Braun's splint would have been recognized long ago. Traction should have one object alone—to prevent recurrent over-riding; alinement should be controlled independently by suitable splintage.

Refracture and Late Bowing.—Failure to continue protection long enough may cause refracture or late bowing. In this series the complication occurred only in the femur, always between the eighth and tenth weeks, and usually as the result of anxiety to avoid stiffness of the knee-joint. After about eight weeks of continuous immobilization early clinical evidence of union was accepted, non-weight-bearing exercise was encouraged, and sometimes traction and the Thomas splint were discarded. Actual weight-bearing had seldom been allowed, the second injury usually being sustained while the patient was still in bed and often despite the protection of a calliper splint. On one occasion a fracture was being tested to determine whether union was sufficiently advanced for mobilization of the knee, and refracture occurred in the course of clinical examination. It is obvious that although some fractures of the shaft of the femur may be clinically firm in eight weeks, this date is unreliable and unsafe. If union has not been quite so rapid and protection is needed for a few weeks longer, attempts to accelerate recovery by early mobilization, or even clinical examination with this object, may have precisely the opposite effect. After refracture, a second term of ten weeks' immobilization is needed; the joint becomes stiffer than if no attempt had been made to prevent stiffness. It may sometimes be permissible to begin cautious mobilization of the knee-joint after eight or ten weeks, provided that the limb is held securely by skin-traction in a Thomas splint with a hinged flexed-knee attachment, and that every precaution is taken to protect the fracture from angulatory strain. But the ultimate range of movement of the joint depends on the severity of primary injury to the muscles of the thigh and the degree of fixation of the quadriceps which results from such injury, from subsequent infection, or from low-grade infection of a supracondylar pin track. The question as to whether the knee-joint is immobilized for eight weeks, ten weeks, or twelve weeks is unimportant. and the fear of joint stiffness should not influence the time of immobilization of the fracture. Union of a

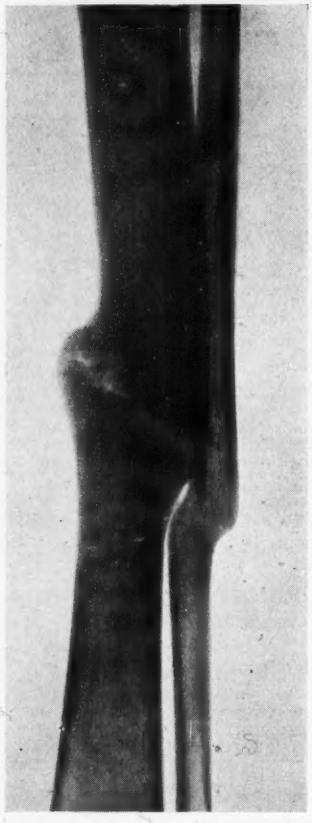

FIG. 288.—Slow union due to interrupted immobilization showing typical heaping up of callus round the fracture (13 months after fracture).

fracture of the mid-shaft of the femur should not be expected in less than about twelve weeks, and even after that, protection of the fracture should be continued by skin traction and a Thomas splint with hinged knee attachment during the first two or three weeks of mobilization of the knee. A calliper splint should seldom be relied upon until there is radiographic evidence of early consolidation and until it is safe to discard the splint in bed at night; its function is to protect the bone from sudden unguarded strain during the fourth and fifth months when weight-bearing activity is increasing rapidly. The patient must not be misled in early days. He should be warned that the minimal periods to be expected are three months complete immobilization, one month protected non-weight-bearing exercise, and one month protected

weight-bearing activity. The importance of early warning and firm insistence was learned in the treatment of air-crews, particularly, for example, during the Battle of Britain, when every pilot was so desperately anxious to return to duty that many employed the skilful art of persuasion in their attempts to be released from bondage.

FRACTURES WITH DISTRACTION

The most serious delaying influence in both closed and infected fractures is distraction of the fragments. Fractures of the tibia which would unite otherwise in about three months, unite after distraction only in six or twelve months. The union of a closed simple fracture is delayed as much by distraction as if it had been converted into an open infected fracture :—

	United by the			
	12th week per cent	*16th week* per cent	*24th week* per cent	*48th week* per cent
Uncomplicated fracture of the tibia	42	70	95	100
Closed fractures of the tibia with distraction	—	6	31	97
Open fractures of the tibia with infection	2	6	35	91

Overpull during the first few hours, before the fracture-hæmatoma is replaced by granulation tissue, appears to be unimportant; but distraction at any later stage inhibits bone formation, promotes fibrous tissue growth, and causes inactivity of the fractured surfaces. The radiographic appearances show characteristic indolence which is in striking contrast with the heaped-up callus formation round the sides of a fracture subjected to interrupted immobilization (compare *Fig.* 288 and *Fig.* 291). Radiography alone suggests that the type of delay is different, and this is confirmed by investigation of the ' undisturbed union time '.

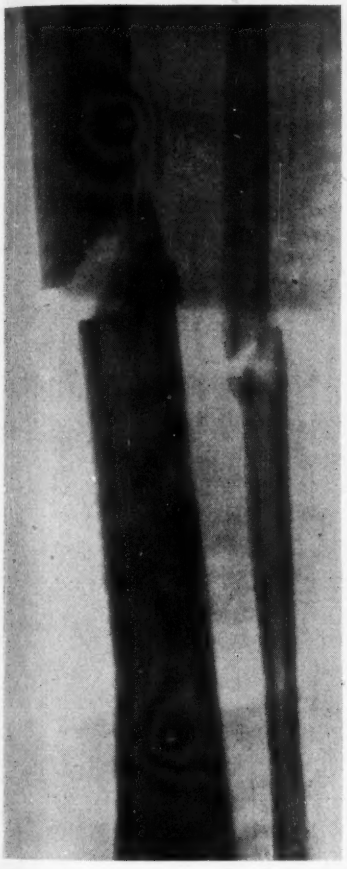

Fig. 289.

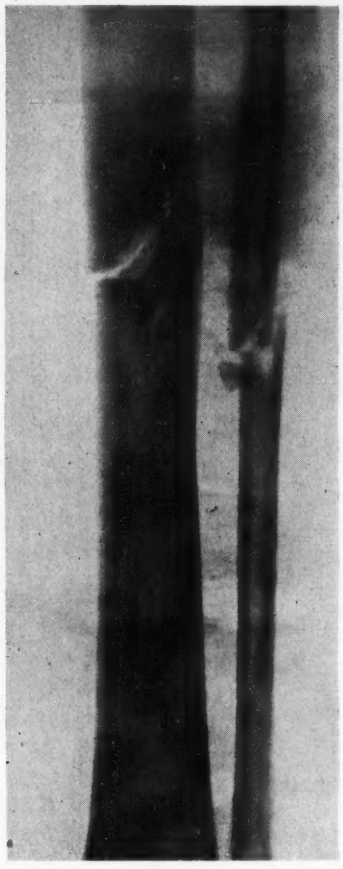

Fig. 290.

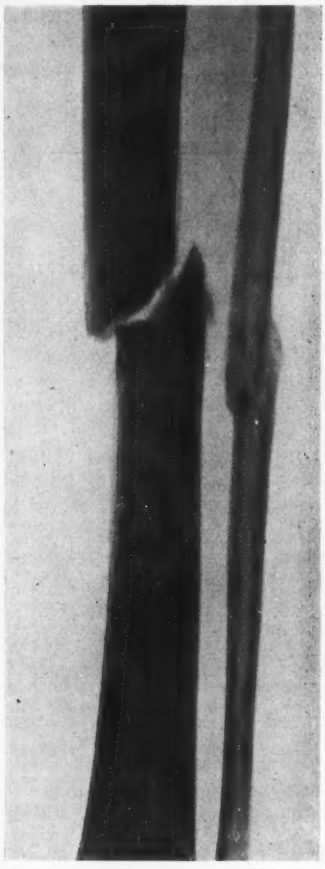

Fig. 291.

FIGS. 289–291.—Slow union due to distraction. Distraction of the fragments (*Fig.* 289) was corrected within 8 days (*Fig.* 290). Nevertheless 10 months later the fracture was still ununited, and the fragments were typically indolent (*Fig.* 291).

Persistent Effect of Corrected Distraction.—By ' undisturbed union time ' we mean the time required for union after cessation of interruption in one series, and after correction of distraction in the other; this has been compared with the time required for union in uncomplicated fractures. The figures in *Table VII* may be summarized as shown overleaf.

It is clear from this analysis that interruption of immobilization is a purely mechanical interference; when interruption ceases, union of

UNDISTURBED UNION TIME IN FRACTURES OF THE TIBIA

	United by the 12*th week* per cent	24*th week* per cent
Uncomplicated fractures	42	95
After interrupted immobilization	36	92
After distraction	14	61

the fracture proceeds almost normally. Distraction, on the other hand, causes delay which persists long after the distraction is corrected. It is not a mechanical problem of bridging a gap; it is a physiological interference; distraction appears to alter the quality of repair and promote fibrous tissue rather than osseous growth. The fact that correction of overpull does not prevent slow union is also shown in the detailed analysis of all distracted fractures in the series (*Fig.* 294). The delaying effect is not proportionate to the length of time during which the distraction persists. It is much the same whether it lasts for a few days, for several weeks, of if it is not corrected at all. Moreover, the delay is the same at whatever stage of repair the distraction occurs. Finally it is the same no matter whether the degree of overpull amounts to a quarter of an inch, a third of an inch, or half an inch.

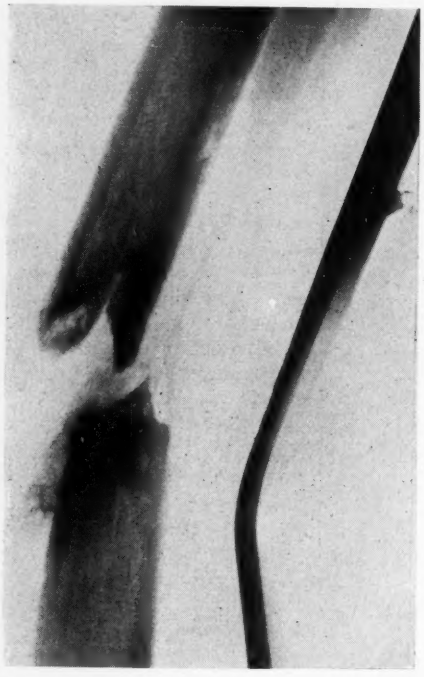

FIG. 292.—Distraction of fracture shaft of femur owing to the use of a suspended weight to control alinement as well as length. Union was very slow. For this reason a Braun's splint is dangerous.

Distraction most Harmful in the Tibia.—Only one feature mitigates the delaying influence of distraction. It is much less harmful in the femur than in the tibia, and it is least harmful in the fibula. Whereas 43 per cent of distracted fractures of the femur united in less than four months, only 6 per cent of distracted tibial fractures united in this time and most of them took nearly twelve months (compare *Tables I* and *II*). Furthermore, when union of fractures of the tibia was strikingly delayed, fractures of the fibula sustained simultaneously, at the same level, and subjected to the same distraction, showed relatively little delay. It appears that the more free the blood-supply of a bone the less harmful is distraction; the less free the blood-supply the greater is the delay; and it is possible that fibrous tissue response is the result of attenuation of blood-vessels in young granulation tissue and deprivation of vascular supply to a bone already feebly supplied with blood.

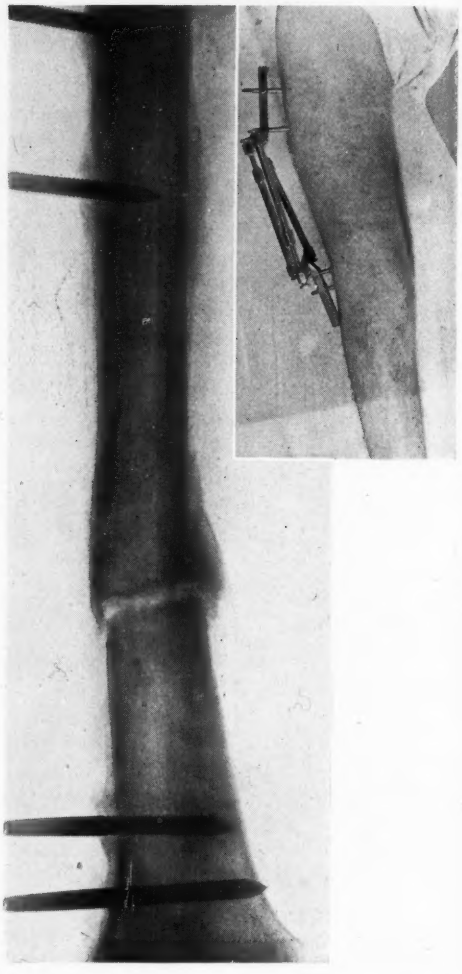

FIG. 293.—Distraction may occur even without a suspended weight. In this case Haines's apparatus has been used (*inset*). Distraction causes serious delay even when the fragments are rigidly immobilized.

Distraction when no Weight is used.—Distraction does not necessarily involve suspension of a weight from a limb. Methods of skeletal transfixion with incorporation of pins in

mechanical devices, which have been described in America, are no less harmful than methods of skeletal traction with suspended weights. Rigid fixation in the distracted position does not nullify the effect of distraction, no matter whether the pins transfixing the fragments are fixed in a Roger Anderson apparatus,[43-45] Haines apparatus,[46] in McKee clips with a Thomas splint,[49] or in a plaster cast.

INFLUENCE OF TRACTION ON THE RATE OF UNION OF FRACTURES

Excessive pull and distraction of fragments can be avoided, but simple traction has long been regarded as an essential feature of the treatment of fractures of the shaft of the femur. Since it is known that overtraction amounting only to a quarter of an inch causes serious

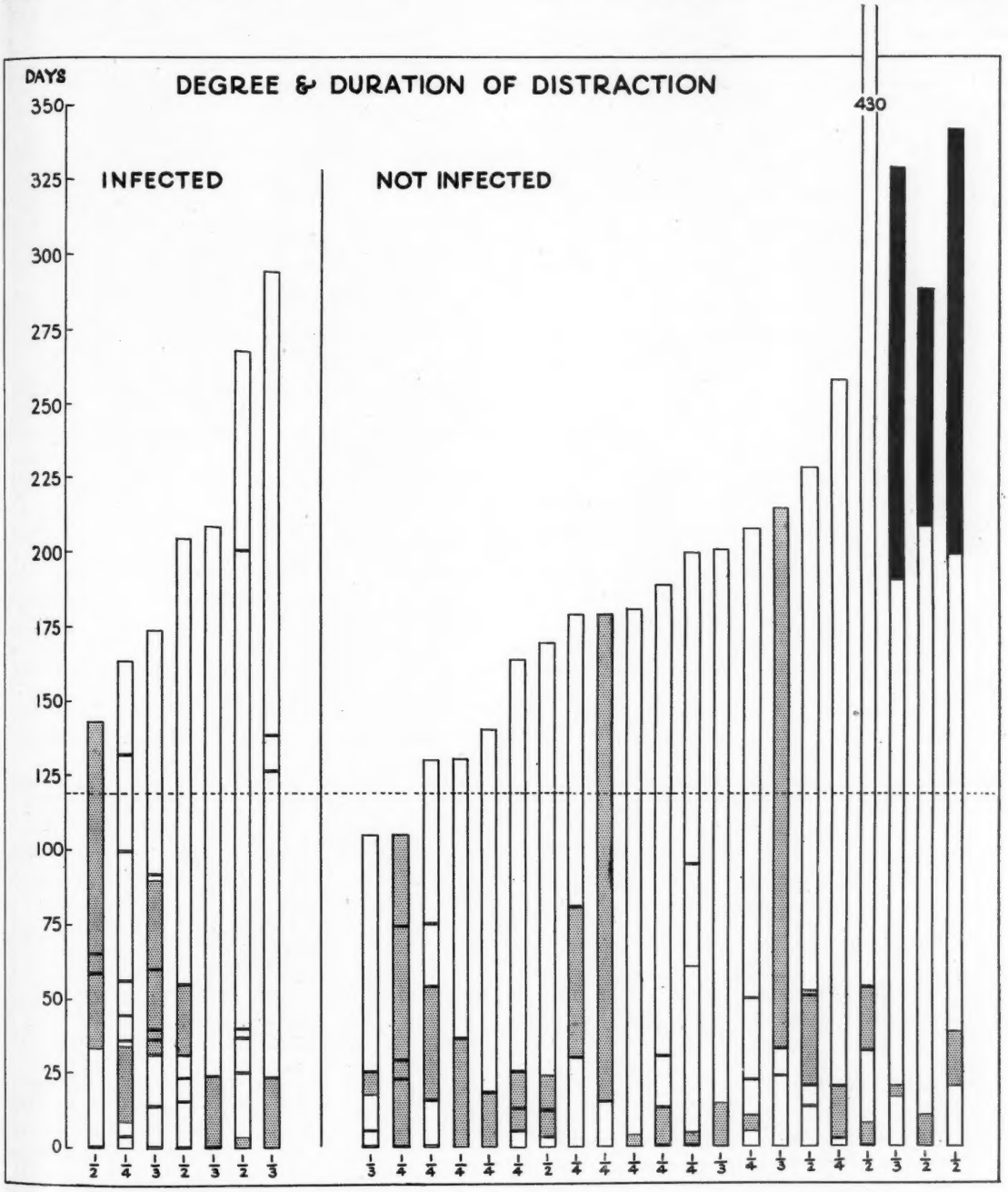

FIG. 294.—*Detailed analysis of all distracted fractures of the tibia.* The time and duration of distraction is shown in stipple. Cross lines indicate remanipulations or other adjustments of position. The degree of distraction in ¼, ⅓, and ½ in. is shown below each column. The horizontal line across the diagram marks the end of the 4th month and gives an indication of the serious degree of delayed union in nearly every case. The delay is independent of time, degree, or duration of overpull. (In the last three cases bone-grafting operations were performed as shown in black.)

delay, is it not probable that any traction causes some delay? In a bone so freely supplied with blood as the femur, where even excessive traction is relatively harmless, minor delay due to simple traction would almost certainly be overlooked. But what of the tibia with its poor blood-supply, in which the effect of excessive traction is so striking? Does simple traction without obvious distraction delay the union of fractures of the tibia? It is only during the last ten or fifteen years that continuous skeletal traction has been used in unstable fractures of the shaft of the tibia. Before that time the fragments were held in splints or plaster in the best possible position, and slight over-riding was often accepted. Nine years ago, even before continuous traction was used, Wardle expressed the opinion that over-riding fractures of the tibia united more rapidly than fractures reduced perfectly by skeletal traction, and this view was confirmed by Eastwood.[50] Analysis of 199 simple fractures of the tibia, one group treated by continuous pin traction on an open splint or by traction from a pin incorporated in plaster, and the other by simple manipulation and plaster, leaves little room for doubt (*Table VIII*). No fracture is included in which there was evidence of actual distraction, but only 22 per cent of fractures treated with traction were united in twelve weeks, whereas 45 per cent of fractures treated without traction united in that time. By the sixteenth week 43 per cent of fractures treated with traction, and 75 per cent of fractures treated without traction, had united. By the twenty-fourth week the influence of simple traction had disappeared, but until then the delay resulting from the use of traction had averaged four weeks. Possibly this delay is to be accepted as the necessary cost of securing more perfect reduction. The cost is not heavy. But is the alternative method of controlling unstable fractures by open reduction and internal fixation associated with the same penalty?

INFLUENCE OF OPEN REDUCTION ON THE RATE OF UNION OF FRACTURES

Until recent years it was believed that operative reduction and the plating of fractures caused slow union or even non-union. The fear of metallic internal fixation was largely dispelled by success in the treatment of fractures of the neck of the femur by the three-flanged nail, and confidence was finally restored by the development of stainless steels, vitallium, and other inert alloys which retained their fixation for many months because bone reaction was minimal. It is now realized that the source of non-union of plated fractures in former days was the false reliance placed on internal fixation, by which surgeons were tempted to minimize or discard external fixation by splints and plaster. When screws and plates became loose in the bone the fracture was not immobilized at all. Reliance can now be placed on the continued fixation of screws and plates which are adequately protected by external splintage or plaster, and it is found that a skilfully performed operative reduction does not delay union. An ill-advised or technically imperfect operation may be disastrous because it often combines in a single case all the delaying factors of interrupted immobilization, stripping of soft tissues, loss of blood-supply, angulation, too early weight-bearing, and infection. But if the operation is performed at the right time, with perfect technique, good after-treatment, and freedom from wound infection, the fracture unites as quickly as by any other method of treatment. The rate of union is the same as in uncomplicated fractures treated conservatively without interruption of immobilization. In fractures of the shaft of the tibia it is more rapid than after methods of continuous traction, and there is a gain of several months as compared with methods of traction which involve distraction. The conclusion must be that although operative internal fixation is associated with certain risks, and is unlikely to replace methods of traction in the treatment of fractures of the shaft of the femur, it is the method of choice in unstable fractures of the shaft of the tibia which are liable to redisplacement. It is not associated with the delay which occurs when these fractures are treated by continuous traction, a method of treatment recently introduced which may well have been "an advance in the wrong direction".

OPEN FRACTURES WITH INFECTION

Infection is not a Cause of Non-union.—The belief that infection is a cause of non-union has survived for many years. Even within the last eighteen months, two surgeons reporting non-union in 25 per cent of infected fractures of the shafts of long bones displayed neither surprise nor regret; they clearly believed that failure in one fracture out of four had been inevitable. The fact is that infection is not a cause of non-union; it is a cause only of delayed union. An infected fracture is exactly like a simple fracture except that every stage of repair is prolonged. Bone is destroyed not only by injury but also by infection; bone is decalcified not only by traumatic hyperæmia but also by infective hyperæmia; in the infected fracture a gap may have to be bridged. Moreover infective hyperæmia may persist for many months, and not until it has subsided can recalcification begin. Throughout this stage, granulation tissue between the bone ends is susceptible to shearing and rotation strain. The effect of interrupted immobilization is even more striking than in the case of closed fractures. Carelessness at a

time when the fracture is being replastered causes secondary fracture of the callus (*Figs.* 295–298), and if interruptions are repeated often enough the fracture fails to unite. On the other hand if complete immobilization is continued without interruption not only until infection is healed, but for several months afterwards until losses are made good and the whole area is recalcified, infected fractures unite with the same certainty as fractures without

Fig. 295.

Fig. 296.

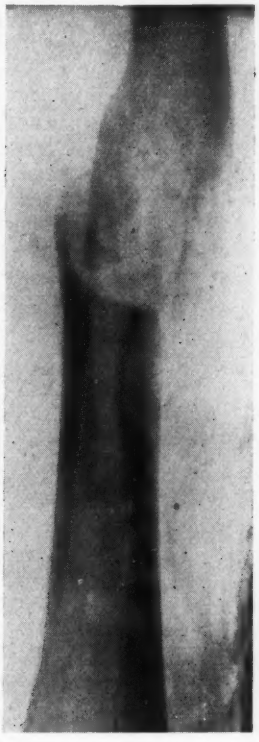

Fig. 297.

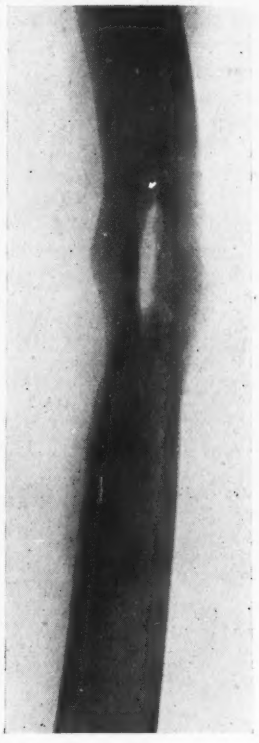

Fig. 298.

FIGS. 295–298.—*Infected fracture shaft of femur with sequestration of the whole thickness of the shaft.* This is the exception to the rule that union should be accelerated by removing sequestra at the first possible moment. When the whole thickness of the shaft is involved, sequestrectomy should be deferred until an involucrum is forming (*Fig.* 295). Recalcification begins as soon as infection is healed by sequestrectomy (*Fig.* 296). Special care must be taken at times of replaster to avoid careless handling which may cause refracture and further delay (*Fig.* 297). Every infected fracture will unite without shortening if immobilization is complete and prolonged, no matter how much bone has been destroyed (*Fig.* 298).

infection. The degree of delay in the union of an open fracture depends almost entirely on the length of time during which infection persists. It is true that open fractures which heal without infection do not unite quite as rapidly as closed fractures (*Table II*). They compare more closely with the group of fractures in which immobilization is interrupted because reduction is difficult. But delay is serious only when there is infection, in which case no more than 6 per cent of fractures unite within sixteen weeks, and only 35 per cent within twenty-four weeks. This delay, due to long-continued infection, is to be avoided by (*a*) skilful primary excision of wounds, (*b*) early sequestrectomy, (*c*) early replacement of destroyed skin by grafting.

Importance of Early Excision of Wound.—It is unnecessary to emphasize the importance of prompt and thorough excision of wounds, of removing dead and devitalized tissue, refraining from suture of the deep layers of the wound, and seldom suturing even the skin. The degree of infection, and therefore the fate of the fracture, is determined within the first few hours after injury. Modern standards will not have been achieved until every wound is properly excised within six or eight hours, until every patient with an open fracture has a temperature chart which is flat, and until all wounds are healed within a few weeks.

Importance of Early Sequestrectomy.—The need for early sequestrectomy calls for emphasis. Sequestration of bone is often ignored, and surgeons expect fractures to unite despite it. Moreover sinuses are allowed to continue discharging because fragments of wood, clothing, or other non-opaque foreign bodies are not removed. But a sequestrum or foreign body causes persistent infection; infection causes

hyperæmic decalcification; decalcification prevents union of the fracture. An infected fracture seldom begins to unite until infection is quiescent, and on the other hand it is often astonishing how rapidly union takes place after many months of indolence when a discharging sinus is healed by removing the sequestrum which was responsible. No other measure is needed than to remove the sequestrum and immobilize the fracture in plaster; the fractured surfaces require no freshening; the bone cavity and the sinus must under no circumstances be scraped; no bone-graft is needed; no plate or screws should be employed. Scraping is harmful because it produces as many new sequestra as it removes. The insertion of a bone-graft is obviously wrong when the purpose of the operation is to remove dead bone. Similarly it is wrong to insert plates and screws.[51] The fact that vitallium is 'inert' and 'silent' in healed wounds does not alter the fact that it is a sequestrum in infected wounds. Even with the assumed protection of chemotherapy, plating of infected fractures is harmful, often dangerous, and always unnecessary.

Importance of Early Skin-grafting.—The delaying influence of infection in the soft tissues round a fracture is even less well recognized than that of infection of the fracture itself. Chronic ulceration over the subcutaneous surface of a fractured tibia is a frequent cause of delayed union. Sometimes after many months of indolence due to persistent low-grade infection, the healing of an overlying chronic ulcer by skin-grafting is followed by union of the fracture almost before the last stage of the skin-grafting operation is completed. The complication should be avoided by early skin-grafting.[52,53] If skin is destroyed it should be replaced by means of a free razor graft at the first possible moment, preferably at the time of the original wound excision, and certainly when the first plaster is changed three or four weeks later. Only in neglected cases should it be necessary to use a whole-thickness direct-transference pedicle skin-flap from the opposite calf.

TABLES FROM A REVIEW OF 804 FRACTURES OF THE SHAFTS OF THE TIBIA AND FEMUR TREATED IN ORTHOPÆDIC CENTRES OF THE ROYAL AIR FORCE AND THE LIVERPOOL ROYAL INFIRMARY

(*Wing Commander W. D. Coltart, A.A.F.*)

Table I.—Summary of Reviewed Cases

		Cases
Fractures investigated in detail:—		
Shaft of tibia		417
Shaft of femur		142
Excluded from detailed investigation:		
Patients under 16 years of age	82	
Bone-grafting to accelerate union	17	
Treatment not concluded	35	
Early records incomplete	83	
Amputations	14	
Deaths	14	
	—	245
		804

Table II.—Time of Union of Fractures of the Shafts of the Tibia and Fibula
(417 *Fractures*)

Weeks for Union	Open						Closed					
	Whole Series		*Infected*		*Not Infected*		*Distraction*		*Interrupted Immobilization*		*Continuous Immobilization*	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
0–12	99	23·7	1	2	4	8·9	—	—	11	10·6	83	41·7
12–16	97	53·7	2	3·8	14	30·4	1	31·3	23	63·5	57	53·7
16–20	77		4	7·7	10	21·7	3		23		37	
20–24	50		11	21·1	5	10·8	1		20		13	
Over 24	94	22·6	34	65·4	13	28·2	11	68·7	27	25·9	9	4·6
Total	417	100	52	100	46	100	16	100	104	100	199	100

Table III.—Time of Union of Fractures of the Shaft of the Femur

(142 *Fractures*)

Weeks for Union	Whole Series		Open		Closed							
			Infected and Uninfected		*Distraction*		*Refracture*		*Open Reduction*		*Uncomplicated Fractures*	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
0–12	54	38·1	4	17·4	4	28·6	—	—	—	—	46	61·4
12–16	27	19·0	3	13·0	2	14·3	1	6·3	2	14·3	19	25·4
16–20	21	14·8	4	17·4	4	28·6	7	43·7	1	7·1	5	6·6
20–24	15	10·5	—	—	3	21·4	5	31·3	6	42·9	1	1·3
Over 24	25	17·6	12	52·2	1	7·1	3	18·7	5	35·7	4	5·3
Total	142	100	23	100	14	16	16	100	14	100	75	—

Table IV.—Effect of Comminution on Rate of Union of Fractures of Shaft of Tibia

(188 *Fractures*)*

Weeks for Union	Non-comminuted		Comminuted	
	Number	Per Cent	Number	Per Cent
0–12	55	40·7	23	43·4
12–16	40	29·6	14	26·4
16–20	27	20·0	8	15·1
20–24	8	5·9	5	9·4
Over 24	5	3·8	3	5·7
Total	135	100	53	100

Table V.—Effect of Site and Shape of Fracture on Rate of Union of Fractures of the Shaft of the Tibia

(135 *Non-comminuted Fractures*)*

Weeks for Union	Spiral		Oblique		Transverse		Total Middle Third		Total Lower Third		Total All Types	
	M. ⅓	L. ⅓	M. ⅓	L. ⅓	M. ⅓	L. ⅓	M. ⅓	Per Cent	L. ⅓	Per Cent	Number	Per Cent
0–12	13	6	12	5	17	2	42	40·8	13	43·6	55	40·7
12–16	7	4	9	6	12	2	28	27·2	12	40·5	75	55·6
16–20	3	2	9	3	8	2	20	19·2	7	15·9		
20–24	2	—	2	—	4	—	8	8	—	—		
Over 24	2	—	2	—	1	—	5	4·8	—	—	5	3·7
Total	27	12	34	14	42	6	103	100	32	100	135	100

* *Tables IV* and *V* analyse the 199 closed fractures of the tibia not complicated by distraction or interrupted immobilization, but since 100 were in the lower third, 103 in the middle third, and only 11 in the upper third, the last group has been excluded as being too small for detailed analyses.

Table VI.—Effect of Weight-bearing and Angulation on Rate of Union of Fractures of the Shaft of the Tibia

(191 *Fractures*)*

Weeks for Union	Weight-bearing in Plaster (191 Fractures)				Angulation with no Weight-bearing (73 Fractures)				Angulation with Weight-bearing (118 Fractures)			
	No Weight-bearing		*Weight-bearing*		*No Angulation*		*Angulation*		*No Angulation*		*Angulation*	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
0–12	37	50·7	44	37·3	30	55·5	7	36·8	31	40·8	13	31·7
12–16	17	23·3	38	32·2	11	20·6	6	31·5	26	34·2	12	29·2
16–20	14	19·2	21	17·8	9	16·6	5	26·5	13	17·1	8	19·5
20–24	2	2·7	9	7·6	1	1·8	1	5·2	5	5·3	4	9·8
Over 24	3	4·1	6	5·1	3	5·5	—	—	2	2·6	4	9·8
Total	73	100	118	100	54	100	19	100	77	100	41	100

* Of the 199 fractures, 8 have been excluded because it was uncertain whether weight-bearing had been allowed or not.

Table VII.—Undisturbed Union Time, Fractures of the Shaft of the Tibia, after Correction of Distraction and Cessation of Interruption, Compared with Union Time in Simple Fractures with Uninterrupted Immobilization

(318 *Fractures*)*

Weeks for Union	After Correction of Distraction		After Cessation of Interrupted Immobilization		Uninterrupted Immobilization	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
0–12	2	14·3	38	36·5	83	41·7
12–24	7	46·7	58	55·8	107	53·7
Over 24	6	40	8	7·7	9	4·6
Total	15	100	104	100	199	100

* This table analyses the 319 closed fractures of the tibia, with the exception of one in which distraction was never corrected (*see Fig.* 294). The other distracted fractures shown in *Fig.* 294 were compound and are therefore excluded from this table.

Table VIII.—Effect of Traction (without Distraction) on Rate of Union of Fractures of the Shaft of the Tibia

(199 *Fractures*)

Weeks for Union	Traction						No Traction	
	Traction on Open Splint		*Pin and Plaster*		*Total*		*Manipulation and Plaster*	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
0–12	1	14·3	6	24	7	21·8	76	45·5
12–16	1	14·3	6	24	7	21·8	50	29·9
16–20	4	57·1	6	24	10	31·3	27	16·2
20–24	1	14·3	5	20	6	18·8	7	4·2
Over 24	—	—	2	8	2	6·3	7	4·2
Total	7	100	25	100	32	100	167	100

SUMMARY AND CONCLUSIONS

1. Uncomplicated fractures treated by simple manipulation and plaster are uniting as quickly to-day as in former years. Fractures of the tibia were never soundly united in six or seven weeks; they were only 'clinically united', and late yielding, mal-union, or non-union often occurred.

2. Ten to twelve weeks should be accepted as the minimal period of immobilization of lower-limb shaft fractures; lengthy protection is not only more safe, but it accelerates functional recovery. Only the *minimal* period of immobilization can be fixed; the 'average period' is meaningless because there is such wide variation between uncomplicated fractures which unite in about 12 weeks, difficult fractures in 12 to 24 weeks, infected and distracted fractures in 6 to 12 months, and avascular fractures in 1 to 3 years.

3. Interrupted immobilization, traction and distraction, infection, persistent angulation, too early weight-bearing, and loss of blood-supply to the fragments cause delayed union; but only when there is failure to prolong the period of immobilization accordingly do they cause non-union. Every fracture unites if it is immobilized long enough.

4. The greater use of skeletal traction accounts for recent increase in the frequency of slow union, particularly in fractures of the shaft of the tibia. The time required for union is trebled, or more than trebled, by distraction, even if it amounts only to ¼ in. and even if it is corrected within a few days. Traction without obvious distraction also causes marked delay in fractures of the tibia.

5. In skilled hands operative reduction does not cause delay, and it is better to prevent redisplacement of unstable fractures of the tibia by internal fixation than by continuous traction. The decision to use internal fixation should be made within a day or two of injury; it should not be a last resort.

6. Early weight-bearing in plaster, in skeletal transfixion apparatus, or in a calliper splint, does not accelerate union; it delays it. The delay is most striking if angulation of the fragments is imperfectly corrected, because weight-bearing then causes distraction on one side of the fracture. Weight-bearing before the stage of clinical union in unwise.

7. Reduction of fractures of the shaft of the femur should be secured immediately by manipulation and not gradually by heavy traction; light continuous traction should then be used only to maintain length and not also to control alinement.

8. The time of immobilization of fracture of the femur should not be influenced by the fear of knee-joint stiffness. Permanent stiffness is due not so much to prolonged immobilization of the joint as to anchorage of the quadriceps by the initial muscle injury, subsequent infection, or low-grade infection of pin tracks.

9. Infection of fractures and of neighbouring soft tissues causes serious delay and should be minimized by early wound excision, early sequestrectomy, and early replacement of destroyed skin by grafting. Infection of a fracture should not be a cause of non-union.

We are grateful to Air-Marshal Sir Harold Whittingham, C.B.E., Director-General Medical Services of the Royal Air Force, for valuable aid in facilitating the investigation of R.A.F. cases and for permission to publish the results. We are also indebted to Mr. J. Nish, First Assistant to the Orthopædic Department of the Liverpool Royal Infirmary, who, despite the heavy pressure of other commitments, investigated and analysed all the Liverpool cases.

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SHORT NOTES OF RARE OR OBSCURE CASES

PELVIC AND PERINEAL TUMOUR SIMULATING PERINEAL HERNIA

BY IAIN MATHESON
SENIOR RESIDENT SURGEON, ST. GILES' HOSPITAL, L.C.C.

PERINEAL hernia is an exceedingly rare form of hernia. A tumour arising in the pelvis and, by extension into the perineum, simulating a perineal hernia is even rarer. The following report of such a case seems worthy of record.

CASE REPORT

The patient, a man of 65, an accountant, was admitted on Feb. 14, 1942, on account of retention of urine. He stated that for the last week he had had difficulty in passing urine, being able to pass only small amounts at a time, and had had complete retention for two days.

FIG. 299.—Photograph showing the extent of the perineal portion of the tumour.

ON EXAMINATION.—He was a man thin almost to the point of emaciation. Apart from the tumours to be described and the retention of urine, there were no abnormal physical signs.

1. Over the posterior aspect of the left shoulder there was a tumour about 5 in. in diameter in the subcutaneous tissue and adherent to the skin, though not to the deeper structures.

2. In the left loin posteriorly there was a tumour about 2 in. in diameter, subcutaneous in situation.

3. Over the 8th left rib in the anterior axillary line was a tumour about ½ in. in diameter, also subcutaneous.

4. There was an enormous tumour in the perineum extending backwards beyond and to the left of the anus and forwards to the pubes (*Fig.* 299). The skin over this tumour was œdematous and freely movable on the tumour. The penis and testicles were quite freely movable in relation to the tumour. The tumour could be rocked slightly from side to side and appeared to be attached deeply, though, owing to its size, the pedicle could not be felt. When the patient coughed, an expansile impulse could be felt in the tumour.

5. There was a tumour about 4 in. in diameter to the right of the anus and apparently situated in and protruding from the right ischiorectal fossa.

6. Rising out of the pelvis half way to the umbilicus was another tumour which at first was thought to be the distended bladder. However, this tumour remained unchanged after 38 oz. of urine had been withdrawn by catheter. This tumour was within the abdomen and could not be moved.

In response to questioning, the patient stated that he had first noticed a swelling in the perineum at least ten years ago, but had never paid much attention to it. He said that at one time the tumour would

disappear, but that it had not done so for a year or two. In the last twelve months the tumour had been increasing in size rapidly, and now chafed his thighs as he walked. He had previously suffered no disability at all. The patient had noticed the tumour on his shoulder, but could not say for how long. He was unaware of the presence of the other tumours.

At first, the large tumour in the perineum was thought to be a perineal hernia, the other tumours being considered lipomata, but, when a cystogram (*Fig.* 300) showed the bladder in the left iliac fossa,

FIG. 300.—Cystogram showing bladder in left iliac fossa.

I concluded that this was a case of dumb-bell tumour partly in the pelvis and partly in the perineum.

PROGRESS.—After the bladder had been emptied by catheterization on admission, the patient found himself unable to pass urine, and a catheter had to be tied in to drain the bladder. The urine contained pus, and he evidently had a severe cystitis, and probably infection of the kidneys. The urine remained dirty despite bladder wash outs, and when the catheter was removed after a week, the patient was still unable to pass urine naturally. The catheter was therefore re-inserted.

It seemed clear that it would be necessary to remove the pelvic and perineal tumour in order to relieve the obstruction to the outflow of urine from the bladder, and to allow the latter to resume its rightful place before the urinary infection could be adequately dealt with.

AT OPERATION.—On Feb. 27, 1942, the operation was performed, with spinal anæsthesia supplemented by nitrous oxide and oxygen.

A midline incision was made from the symphysis pubis to the umbilicus and a soft mass was found rising out of the pelvis half-way to the umbilicus and pushing the peritoneum upwards. The peritoneal cavity was opened, and, after it had been established that the tumour was extraperitoneal, was closed and the peritoneum stripped off the upper part of the tumour. The tumour felt cystic and aspiration was attempted, but with no success. The tumour fitted the pelvis so tightly that it had to be removed by subcapsular enucleation, and it was found to be lobulated and soft. The tumour appeared to have a pedicle in the left ischiorectal region, but this tore across very easily without any hæmorrhage being caused. The capsule was next examined and was found to be densely adherent to the prostate and urethra in the left iliac fossa. As there appeared to be great danger of damage to these structures and to the ureters (their position was never determined) if the adherent parts of the capsule were cut away, only that part which had stripped easily was removed. The cavity in the pelvis was packed with gauze to check oozing and the wound closed.

The patient, who had hitherto been in the Trendelenburg position, was now put in the lithotomy position and an incision made more or less in the midline over the perineal tumour. The skin and subcutaneous tissues were very œdematous, but the tumour was easily reached and shelled out. As expected, it was attached in the region of the left ischiorectal fossa. Hæmostasis was secured and the wound sewn up with a tube drain.

The tumour removed from the abdomen was yellowish-grey in colour, lobulated, and very soft and œdematous. It weighed 20 oz. The tumour from the perineum was larger and more vascular and its capsule was adherent in places. It was yellowish-grey in colour and weighed 37 oz.

The patient's condition at the end of the operation was poor and a drip-blood transfusion was started. However, despite some initial improvement, he never picked up, and, gradually getting weaker, died on March 4.

POST-MORTEM EXAMINATION.—Autopsy revealed severe cystitis and pyelonephritis. Subcutaneous tumours were found in the positions described above. The tumour on the right side of the anus had a lobe which filled up the right ischiorectal fossa and extended above the levator ani muscle on the right. The bladder was about half-way between its position at operation, in the left iliac fossa, and its normal position. Part of the tumour capsule was adherent to the prostate, which was riddled with small nodules of growth. On the left of the bladder was a tumour oval in shape and 4 in. long, the presence of which had hitherto been unsuspected.

Microscopical examination of the tumour removed at operation showed a very œdematous, degenerating, cellular fibroleiomyoma, possibly sarcomatous of low-grade malignancy. Examination of the tumours removed post mortem showed that the two tumours in the pelvis were fibroleiomyosarcomata, that the prostate and base of the bladder were invaded by secondary sarcoma, and that the fatty tumours on the shoulder and back showed secondary sarcomatous infiltration.

DISCUSSION

It is difficult to know whether to be more surprised at the lack of symptoms, prior to the retention of urine, associated with this huge tumour, or at the indifference shown by a comparatively well-educated man to the presence of the tumour in a most inconvenient place.

I have been able to find only one similar case reported. In March, 1909, Mr. Lawrie McGavin[1] showed at a meeting of the Clinical Section of the Royal Society of Medicine a woman, aged 36, who had a swelling in the perineum appearing through the left ischiorectal fossa. This was diagnosed as a perineal hernia owing to its easy and complete reduction and expansile impulse on coughing. In the following month Mr. McGavin reported the result of operation in this

case. He had performed a laparotomy and found that there was a tumour completely extraperitoneal and occupying almost the whole pelvis and extending down to the left of the recto-vaginal septum. The tumour was removed with difficulty and found to be continuous with the perineal swelling. It was œdematous and almost diffluent, weighing 3½ lb. Microscopical examination showed it to be a myxoma.

I wish to thank Dr. W. Allen Daley, Medical Officer of Health, London County Council, for giving me permission to publish the notes of this case.

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TRAUMATIC PERFORATION OF THE LARGE INTESTINE

By MAX GREENBERG

SENIOR SURGEON, CHILDREN'S HOSPITAL, JOHANNESBURG

CASE REPORT

A CHILD aged 9, was admitted to the hospital in a collapsed condition and with a wound over the lower abdomen as the result of a portion of a brick wall falling on the abdomen. The skin lesion was one of laceration and extended transversely about 3 in. above the pubis. Catheterization of the bladder produced blood-stained urine. There was general rigidity of the recti. Blood transfusion and immediate operation was decided upon.

AT OPERATION.—The abdomen was opened by a left paramedian incision. Free fluid of a brownish colour was present in the abdominal cavity, and also a few small pieces of solid fæcal matter. There was an impaction of solid fæces in the rectum and sigmoid, and extending well up the descending colon. At the edge of this hard fæcal mass was found a transverse rupture through all layers of the descending colon. The extent of the rupture was less than an inch. On handling the bowel fluid exuded through the opening. The opening was closed and some of the exuded fluid mopped up. The left flank was drained through a separate opening. Sulphonamide powder was left in the abdominal cavity and the wound closed. The lacerated wound was suitably dealt with. Both wounds healed by first intention. The rectum and sigmoid were cleared by olive-oil enemata.

PROGRESS.—The lad made an uninterrupted recovery.

Comment.—By combating the shock by blood transfusion and warmth, immediate operation was possible. Some dullness and maximum rigidity and pain in the left iliac region prompted the site of incision. There was a minimum amount of bowel handling. Sulphonamide was used intra-abdominally and later orally. Both wounds were treated with a combination of sulphonamide and iodoform cream (iodoform, vaseline, and liquid paraffin).

PRIMARY ECHINOCOCCAL CYST OF THE UTERUS

By G. F. LANGLEY, IPSWICH

THE extreme rarity of echinococcal cyst of the uterus merits the publication of the following case in which an intracranial hydatid cyst had been previously excised.

The first case published in North America was described by Welcker et al.[1] in October, 1940, and these authors collected 42 other cases from the literature. Only 1 case (Bull[2]) has been recorded in an English journal as far as could be ascertained. No other cases have been published in the available literature since 1940. Of the 43 cases hitherto published most of the uterine implantations have been found to be secondary deposits from a primary cyst elsewhere in the peritoneal cavity.

CASE REPORT

Miss V. H., aged 31 years, was admitted to the Borough General Hospital, Ipswich, on April 24, 1940, complaining of severe, intermittent pains in the lower abdomen for the past five days. During this time there had been constipation and difficulty in micturition without frequency, but there had been no nausea or vomiting. Anorexia and increasing constipation had been noticed during the previous five weeks.

Menstruation had commenced at the age of 15 years; her periods had always been irregular, occurring for three or four days every six to eight weeks. The loss had always been scanty and there was some dysmenorrhœa. There had never been any vaginal discharge or pregnancy.

The previous history was interesting. In 1923 her home doctor had opened a "cold abscess" behind her right ear, but headache, vomiting, and "the closing of one eye" persisted and a further operation upon the right mastoid was performed at the Cardiff Royal Infirmary. Information kindly supplied from that hospital states that at this operation an extradural hydatid cyst was found and removed. Severe papillœdema was discovered. When seen again at that hospital in 1928 she was found to have bilateral, secondary optic atrophy, but no other lesion of the central nervous system was discovered. She had been blind since that time and had been evacuated to Ipswich from her home in London.

She had never been out of England and had lived only in South Wales and London.

She was an intelligent, rather frail woman; her right eye was totally blind, but she could distinguish large objects of light colour with the left. Extensive optic atrophy of both discs was found, but there was no other lesion of the central nervous system. There was some fullness of the suprapubic region of the abdomen and on rectal examination a tense, semi-cystic swelling was found to be completely filling the pelvic cavity. Differential diagnosis lay between an impacted ovarian cyst and a degenerating cervical fibroid; the absence of menorrhagia supported the former diagnosis.

OPERATION (April 26).—Laparotomy under general anæsthesia revealed a densely adherent mass filling the pelvic cavity; the small uterus and adnexæ were perched on the summit of the mass. After separating adhesions three rounded masses were seen to project backwards into the pouch of Douglas to be adherent to the rectum. The mass now appeared to be a degenerating cervical fibroid. After clamping the right ovarian pedicle an attempt was made to work downwards between the layers of the right broad ligament, but, in dividing the adhesions, the right ureter was severed and was temporarily ligated.

Approach was then made down the left side of the pelvis; the left ovary was allowed to remain and, after many difficulties due to the dense adhesions, the vaginal vault was reached and divided between clamps. Working upwards from the vagina the separation in the right side of the pelvis was completed. The vagina was sutured and the divided ureter was anastomosed by van Hook's method. A corrugated rubber drain was left near to the sutured ureter and a routine closure was performed after general inspection of the abdomen, for by this time the nature of the mass had been ascertained.

SUBSEQUENT PROGRESS. — The patient's progress was stormy. On April 29, she had considerable paralytic ileus which required continuous gastric syphonage and intravenous saline infusion for three days.

On May 7 a profuse serosanguinous discharge from the wound and from the vagina occurred, and the upper 3 in. of the incision broke down, to be held only by the tension sutures. Vomiting recurred and further gastric syphonage was necessary before a repair under local and gas-oxygen anæsthesia was possible.

Until May 9 urine had been passed normally, but on this date urine leaked from the vagina and a fæcal fistula through the abdominal wound added its troubles. Blood transfusions improved her general condition and the fæcal fistula had healed by June 11; a thin brownish vaginal discharge persisted and urine continued to leak from the vagina and abdominal wall.

Cystoscopy on July 27 showed normal functioning of the left kidney but no efflux from the right side, in which a ureteric catheter was obstructed 3 cm. from its orifice.

By Aug. 4 the abdominal wound had healed, but the uretero-vaginal fistula persisted. The general health was now good and after routine investigations a right nephrectomy was performed under spinal anæsthesia on Sept. 6. The excised kidney showed extensive hydronephrosis. Subsequent convalescence was normal and no further leakage of urine occurred. At this stage she left my care owing to the evacuation of the hospital.

At operation no evidence of any other hydatid cyst was found and subsequent radiological examination of the skull, thorax, and abdomen showed no evidence of hydatid disease. No Casoni test was performed. The patient has written me several times and has remained well; on March 2, 1942, she writes that she is able to carry on her normal work in a blind institution and suffers from no abdominal or nervous symptoms.

EXAMINATION OF THE SPECIMEN (*Fig.* 301).—Only after its removal was the actual nature of the cyst detected. The cyst was surrounded by a very tough fibrous capsule through which bulged three smaller

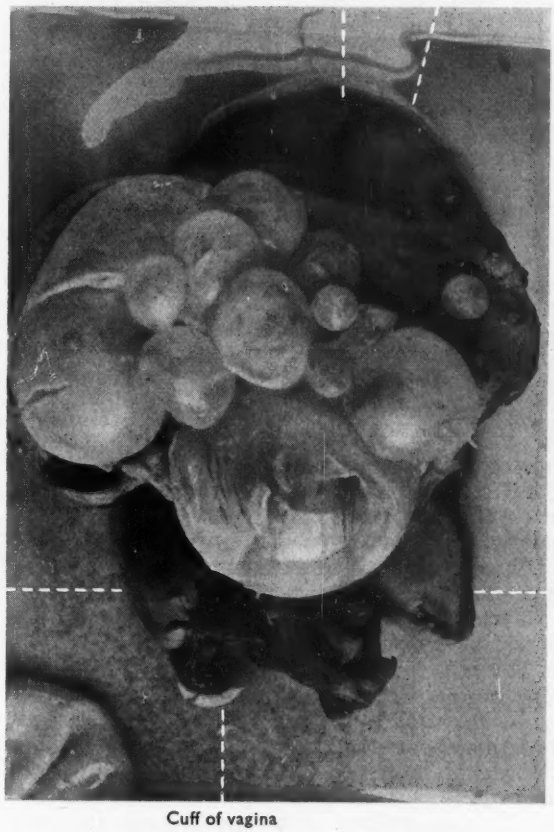

FIG. 301.—Hydatid cyst of isthmus of uterus. The wall of the cyst has been opened, some of the daughter cysts have been removed. The cuff of vagina and cervical portion of the cervix below the cyst are shown. The fundus of the uterus is unopened.

cysts surrounded by less fibrosis. The main cyst was about 6 in. in diameter. The uterus, both tubes, and the right ovary projected from the top of the fibrous capsule and the isthmus could not be separately identified from the capsule. It appeared that the cyst was growing in the uterine wall at the level of the isthmus, stretching the cervix and distorting the uterine canal. When opened, the cyst contained over 80 pearly-white daughter cysts varying in size from 1 mm. to 10 cm. in diameter. The lining of the mother cyst was of the same colour and many further broods of daughter cysts were adherent to its wall. The cysts were of the typical hydatid nature and on microscopical examination showed the characteristic hooklets. No calcification or infection of the cyst was present.

DISCUSSION

Interesting features of this case are the unusual sites of the two hydatid cysts. The original cyst gave rise to symptoms of cerebral compression in 1923, resulting in papillœdema and optic atrophy. No other lesion of the central nervous system ever developed and the cerebral compression was relieved by the removal of the cyst, but this was, unfortunately, too late to save the sight.

Primary hydatid cysts of the brain are rare.[3,4] In a large series of cases of hydatid disease, collected irrespective of age, the percentage of brain cyst has been 2·1; in patients under the age of 15 years brain cysts are at least seven times as frequent as in adults. Dew[5] states that the explanation of this striking clinical fact is that, in the majority of cases, the hydatid infestation occurs in childhood and owing to the resistant nature of the skull the condition gives rise to grave symptoms and is incompatible with long life. This same authority also states that the right side of the brain is more commonly affected than the left, that the parietal lobe is the commonest site affected, and that the cyst is usually single. The cerebellum is very rarely affected. The latency of cerebral hydatid disease is remarkable, partly due to the slow growth of the parasite and to the simultaneous relative increase in the capacity of the child's cranium. Focal cerebral signs are uncommon and general cerebral symptoms predominate, leading to papillœdema and optic atrophy; according to Thomas[4] the average duration of life after the appearance of the first symptom is one year.

Secondary hydatid cysts of the brain are almost always multiple; the primary is usually in the left side of the heart (Dévé[6]), and the condition is incompatible with long life.

In the case now reported it is thus obvious that there was a primary cyst of the meninges for which relatively early treatment had been achieved.

After an interval of seventeen years the only other cyst found was in the uterus, and a full investigation failed to reveal any other implantations. The uterine cyst may be regarded as a primary abdominal infection, and at the operation no other intra-abdominal implantations were discovered. It is, therefore, interesting to speculate upon the date of the original infection of the uterine wall in which this cyst developed. Either a simultaneous infection of the brain and uterus occurred a few years before the age of 14, or, alternatively, a secondary infestation of the uterus has occurred from the primary focus in the brain. The latency of the uterine condition is remarkable, but may be related to the coincident slow growth of both the cyst and its host, as has already been discussed in regard to cerebral infestation in childhood. Eventually the bulk of the cyst caused its impaction in the pelvis, with the production of pressure symptoms and without any focal symptoms directly related to uterine function.

SUMMARY

1. A case of primary hydatid cyst of the uterus has been described. No evidence of any coexisting hydatid cyst was discovered.

2. A pre-existing hydatid cyst, causing cerebral compression and blindness, had been removed seventeen years previously from the mastoid region.

3. The patient has recovered from a series of serious post-operative complications and has made a complete recovery apart from the blindness caused by the intracranial cyst.

4. A brief discussion of salient features of hydatid diseases affecting two rare sites in one patient has been given.

My sincere thanks are rendered to Dr. Eric Biddle, Pathologist to the East Suffolk Hospital, Ipswich, for the pathological reports and photographs. I also express my gratitude to Dr. J. J. Dubash, E.M.S. Pathologist at the Borough General Hospital, for his assistance with the literature.

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A CASE OF 'IMPASSABLE' STRICTURE DUE TO SECONDARY METASTASES IN PENILE PORTION OF URETHRA

BY ALEC SINGER AND R. Y. DAWBARN
WALTON CITY HOSPITAL, LIVERPOOL

THE following case is interesting because of its rarity.

CASE REPORT

A male, aged 66 years, married, gave a history of six-months' difficulty with micturition. He had also had six-months' hæmaturia, but was not quite sure whether the blood came before micturition, but was certain of its being intimately mixed with the urine. He had to get up about six times during the night, but was not unduly bothered during the day. He had lost one stone during the last six months. On

admission he stated that he had passed no urine that day (Sept. 5), and that for the last month he had increasing difficulty in passing urine.

ON ADMISSION.—Temperature 97·4°; pulse 72, and of poor volume. He was pale and his general condition was very poor.

Abdominal examination showed the bladder up to the level of the umbilicus. Nothing else significant.

Rectal examination: The prostate was hard and "knobly", enlarged to about twice normal, and obviously malignant.

A diagnosis of acute retention due to carcinoma of the prostate was made. An attempt was then made to catheterize him, but two hard masses were felt in the penile portion of the urethra. The more distal was just above the glans penis, whilst the more proximal was an inch above the former. Catheters could be passed beyond the first, but no instrument could be passed beyond the second nodule. Because of the marked induration it was thought that these were metastases and that the more proximal had caused a malignant stricture. A suprapubic cystotomy under local 1 per cent novocain was done the same night, and at operation a hard mass was felt involving the base and neck of the bladder.

The patient died four days later (Sept. 9) of uræmia.

POST-MORTEM REPORT.—Post-mortem examination showed a large cancer of the prostate. It was fixed to surrounding parts and spread upwards into the bladder wall. There was pyelonephritis on both sides and metastases in the lymphatic glands, liver, and both pleuræ. Examination of the penis showed a ring stricture caused by the proximal of the two nodules. A section was cut and is shown in the accompanying photomicrograph (*Fig.* 302). The

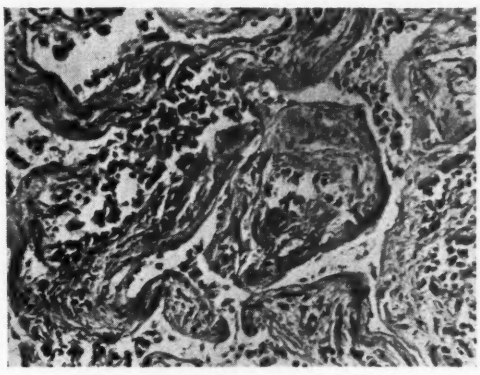

FIG. 302.—Corpus cavernosum penis showing blood-sinuses containing carcinoma cells. (× 66.)

blood-sinuses of the corpus cavernosum are extensively infiltrated with cancer cells.

In the literature available to us at the present time, we have been unable to find any references to metastases in the penile urethra causing complete occlusion due to a malignant ring stricture.

We wish to thank Dr. H. H. MacWilliam, Medical Superintendent, for permission to publish this case.

EDITORIAL

ANGLO-AMERICAN SURGERY

THE Editorial Committee has pleasure in announcing that the distinguished surgeon, Colonel Elliott C. Cutler, Professor of Surgery of Harvard University and now Chief Consultant in Surgery to the United States Forces in this country, has accepted a seat on the Editorial Committee of this JOURNAL.

Colonel Cutler's personality and professional status witness his claim to this position, but he has also the key to our hearts as the disciple and direct surgical heir to that outstanding man and pillar of Anglo-American surgery, Harvey Cushing.

This addition to our staff is welcome in itself, but there is a far deeper meaning underlying the event.

Up till now the BRITISH JOURNAL OF SURGERY has been kept for the writings of British surgeons and the management has been wholly in the hands of the British. Colonel Cutler is the first and only non-British surgeon who has been placed on the committee. His election to this post is a symbol—a symbol of the comradeship and brotherhood which unites our countries in the mighty struggle we are waging for the freedom of the world.

We have all the more pleasure in taking this step as it is in accord with the feelings and aspirations of one who, as Chairman of the Editorial Committee, presided over the policy of the JOURNAL for twenty-one years. Lord Moynihan, among his many accomplishments, did incomparable service in bringing together the surgeons of this country and North America. He would have been the first to welcome with us this representative of our ally as a step towards the high goal of our great endeavour.

PATHOLOGICAL SPECIMENS OF WAR INJURIES

ALMOST a quarter of a century ago the courtesy of the BRITISH JOURNAL OF SURGERY was extended to my former colleague and chief, Sir John Bland-Sutton, who had been charged by the War Office some years earlier with the task of organizing a collection of specimens demonstrating the effects of the projectiles and weapons of war on the bodies of those injured in battle, and of

having such specimens appropriately prepared, preserved, and mounted. In an introductory article to the July number of the BRITISH JOURNAL OF SURGERY, 1919, Sir John was able to write exultingly, and in his own inimitable manner, of the wealth of material that had been accumulated in the conflict of 1914–18 by the enthusiasm and devotion of zealous men interested in surgical pathology.

Through the kind offices of the BRITISH JOURNAL OF SURGERY I am to-day enabled to appeal to *surgeons everywhere* to help in building up once more a National War Collection. Many months ago the Medical Research Council accepted a request from the War Office and the Royal College of Surgeons of England that they should lend their aid in organizing and establishing a comprehensive National Collection of Pathological Specimens illustrating the wounds, injuries, and diseases of 'total' war. The need for co-ordinated effort to this end was regrettably emphasized in May, 1941, when almost the entire Army Medical War Collection which was housed within the precincts of the Royal College of Surgeons of England was destroyed in the holocaust and cataclysm which overwhelmed the Hunterian Museum and its priceless contents, as the result of enemy action. It is envisaged that the new collection now proposed will provide for the future needs in this respect of each of the Services.

A very special duty, therefore, devolves upon our profession: specimens, pictures, photographs, and radiographs illustrating the lesions produced by missiles, war gas, or explosion gas, by secondary missiles such as fragments of masonry, mud, or other debris, and by crushing injury, are required. The effects of the impact of blast-wave in air or water due to high-explosive detonation, and specimens of burns resulting from any form of enemy action demand representation in such a Collection. Preparations, illustrations, or photographs depicting the mode of repair, the consequences of infection, and the results of the treatment of war wounds and injuries are also desired.

Furthermore, specimens are solicited to replace those illustrating injuries sustained in the last Armageddon and also in previous campaigns, which were destroyed in the Royal College of Surgeons' disaster.

The value of any pathological specimen when mounted depends upon a discerning choice of material, upon the care taken in collection, and upon the preliminary fixation applied. A memorandum was therefore drafted by the Sub-Committee on Pathological Specimens outlining the type of material wanted and suggesting suitable methods of preliminary treatment. Copies of this memorandum were distributed widely to various E.M.S. officials and to selected medical officers and hospitals of the Fighting Services; the needs of the Sub-Committee were also promulgated through the medical press and in the *Bulletin of War Medicine*, January, 1942. The response to this appeal has been indeed meagre. If the "magna imago" of John Hunter could return to earth, he would indeed be puzzled and amazed at this paucity of pathological trophies representing over three years of warfare.

It is true that museum material has been collected in the Middle East, and that in this country the aid of colour photography has been employed by certain surgeons; indeed, nearly one thousand specimens of the photographer's art are available for the selectors. The offer by the Metal Box Co., Ltd., of the services of Mr. P. G. Hennell, of their staff, to take coloured photographs by a new process was gratefully accepted by the Medical Research Council; his photographic records of wounds, burns, and the natural processes of repair, and of operative interference, are faithful and exquisite representations of the originals.

It became apparent to the Sub-Committee on Pathological Specimens that something further was required than to rely merely upon the goodwill of surgeons and pathologists in sending material to the collection centres which had been arranged. The need seemed to be established for an enthusiastic, energetic pathologist who would tour the country region by region, gathering specimens and personally securing the co-operation of surgeons and pathologists, and stimulating them to collect specimens against her next visit. Such a pathologist should have an entrée to all medical units, including those of the Fighting Services in this country, in the search for material. The Sub-Committee has been fortunate in securing the interest and enlisting the services of Dr. Joan M. Ross for this task.

As Chairman of this Sub-Committee, *I am appealing to surgeons everywhere* who are brought into contact with casualties or diseases peculiar to 'total' war to remember the needs of our National War Collection, and to co-operate to the fullest possible extent with Dr. Joan Ross in her task of collection. Her address is Miss Joan Ross, M.D., Science Block, Mill Hill Emergency Hospital, London, N.W.7.

It is important to have abundance of material from which to make final choice of specimens for permanent preservation. One day, surgeons and pathological experts with a magpie shrewdness will ultimately pick out the gems and the glittering jewels from amongst those which are less valuable. The Sub-Committee realizes that it will have to make large drafts on the kindness of its friends, but only by personal effort can the altar-lamp of surgical pathology be kept burning.

Nil sine magna
Vita labore dedit mortalibus.

(Nothing is granted to mortals without great labour.)

Remember at all times the needs of the National War Collection and give of your best!

G. GORDON-TAYLOR.

RUBBER GLOVES IN SURGERY

For many years rubber gloves, along with sterile gowns, caps, and masks, have come to be regarded as part of the routine armament of surgeons. To-day when rubber is scarce and gloves in short supply the prospect of being deprived of part of his panoply will be disturbing to many an operator.

The history of the introduction of surgical gloves and the questions that perplexed surgeons in the early days are not without present interest, and may help to dispel some of the alarm. According to the Surgeon-General's catalogue, as early as 1701, one Nicolai, wrote a disquisition upon the use and abuse of gloves, but it was not until 1889 that Halsted began to use them when operating. As late as 1898 surgeons were found to condemn their use. R. T. Morris in the U.S.A. in that year said, " surgeons who were doing first-class work three years ago seem to me to be doing second- or third-rate work now, on account of the interference made by their gloves ". It was only in the same year, 1898, at the German Congress of Surgery, that the question was substantially settled. Mikulicz, Perthes, Döderlein, Zöge von Manteuffel, with others, came to the conclusion that so soon as impermeable gloves, pliable, thin, easily sterilized, and not too costly could be produced, surgeons would certainly use them. Early objections to the routine use of gloves arose chiefly because it was demonstrated that fabric gloves were no protection at all as soon as they were wetted—from within or without—and that the rubber or rubberized gloves available were clumsy enough to interfere seriously with tactile sensation and digital dexterity. Attempts were made to render silk and other fabric gloves impermeable by impregnating or covering them with paraffin or rubber, and to substitute for gloves a coating of similar substances on the skin of the hands themselves.

The danger of rubber gloves also was recognized; organisms too deep in the skin to be reached by any form of cleansing, even with the addition of antiseptics, however powerful, might be sweated into the gloves during the operation and released into the wound by needle puncture. Indeed, it was thought that, puncture being common, gloves might even prove a danger rather than a safeguard. Discussion arose as to who ought to wear gloves. Some held that the surgeon might keep his hands bare if his assistants were gloved. This, apparently, was because it was thought the experienced would be more careful and thorough than the neophyte in cleansing his hands. In order to preserve the sterility of the exterior of the gloves they were sometimes prepared with an outer removable hood or sheath of cloth and laid out in a sterile towel, so that in putting them on, the surface remained untouched. For a considerable time in many clinics, the gloves were boiled and put on full of an antiseptic lotion, generally biniodide or perchloride of mercury, but the objections were recognized, and dry sterilization became the rule.

At first it was agreed that the function of impermeable gloves was to preserve the hands from gross contamination; they should be worn when dealing with any infected case. Only later did it become the accepted routine to wear them for every operation.

Surgical gloves are, in fact, a comparatively recent introduction; they were brought into use when clean surgery had already largely abolished the bugbear of sepsis, and generally speaking can be regarded rather as a refinement of technique than an essential. There were surgeons of the first rank both in technique and results, not long passed away, who never wore gloves in spite of being surrounded by colleagues who did; these men were not die-hards; they believed that gentleness and a dry wound were the best means of obtaining healing by first intention. Though individuals have announced a decrease in the number of cases that failed so to unite, no one has produced significant figures to prove that results have been substantially improved by the use of rubber gloves. A great deal is attributable to the non-touch technique so largely associated with the name of Arbuthnot Lane.

The potential danger of rubber gloves earlier touched upon, is not always fully recognized. The frequency of puncture is higher than generally suspected. The liability of gloves to perforation is shown by the following table. It was made by the Theatre Sister of a Sector Hospital of 900 beds. The numbers refer to individual gloves.

LIABILITY TO PERFORATION OF RUBBER GLOVES

	Gloves used	*Gloves pierced*	
Monday	54	11	
Tuesday	40	22	
Wednesday	55	8	
Thursday	101	18	
Friday	103	16	
Saturday	16	2	
Total	349	77	= 22 per cent

The question of the use of gloves was discussed at a recent meeting of the War Wounds Committee of the Medical Research Council, and the following points were agreed :—

1. That rubber gloves should always be used for operations in septic cases, and by surgeons who were carriers of virulent staphylococci.

2. That surgeons who proposed to operate without rubber gloves should have their hands tested periodically, to make sure that they do not become carriers of virulent organisms.

3. That fabric gloves were undesirable.

4. That surgeons proposing to operate without gloves should endeavour to maintain a 'no-touch' technique as far as possible.

5. That the use of one pair of gloves for several operations, with cleansing of the gloves on the hands between, was undesirable, owing to risk of unnoticed glove punctures in septic cases.

6. That the use of rubber gloves by the theatre sister could in many cases be obviated: (*a*) if forceps were used to pass instruments to the surgeon; (*b*) if the surgeon or the 'first assistant' himself picked up the instruments and either laid them on a special towel or tray after use (with the handles in such a position that they could not be contaminated) or returned them direct to a sterilizer; (*c*) if the needles likely to be needed for an operation were threaded with forceps, or, alternatively, were threaded before they were sterilized.

7. That the use of rubber gloves for surgical dressings is generally unnecessary except in the case of procedures which are too complicated to be managed with forceps alone (*vide* 'The prevention of hospital infection of wounds', *M.R.C. War Memorandum, No.* 6).

At the same Committee, for the preliminary cleansing of the surgeon's hands "C.T.A.B." was mentioned. A surgeon on the staff of one of the teaching hospitals sends the following note:

"*C.T.A.B.*, 1 per cent solution.

"I have used nothing else for the past three months for pre-operative skin preparation. There has been no dermatitis. We take care to avoid soap or any alkaline lotion beforehand. I have used it on my own hands for over two months since I have not been wearing gloves. Repeated applications throughout every operating session have not damaged my skin."

He adds: "I have been without gloves, except in dirty cases, for over two months. The Sister assisting me has been without gloves for the past three weeks. Thus far there is no evidence of harm from lack of gloves.

"Although I support the contention that fingers should be kept out of wounds, yet when handling is unavoidable in the deeper parts of the body (splanchnic neurectomy, nephrectomy, gastrectomy, and colectomy), bare hands can be used harmlessly. I have done many thyroid and breast cases without untoward results."

Nothing in this note must be taken as advocating the abandonment of gloves in operating in times of peace. If, however, despite the production of synthetic rubber gloves in the U.S.A., supply is restricted, plans for meeting the situation can be prepared and no fears need fill the breasts of those who have never known a gloveless régime.

C.W.
E.R.C.

REVIEWS AND NOTICES OF BOOKS

The Surgery of the Ambulatory Patient. By L. KRAFER FERGUSON, A.B., M.D., F.A.C.S., Assistant Professor of Surgery, University of Pennsylvania; with a Section on Fractures by LOUIS KAPLAN, A.B., M.D., F.A.C.S., Associate in Surgery, University of Pennsylvania. 9 × 6 in. Pp. 923 + xvi, with 645 illustrations. 1942. Philadelphia and London: J. B. Lippincott Co. 63s. net.

THIS book of unusual title conjures interesting reflections. What is the 'ambulatory patient' and in what sense does surgery upon such a person differ from surgery performed upon the non-ambulatory patient? A first survey of this large book gives the impression that the author has detailed every conceivable operation which could be done upon a patient and at the same time permit him to walk home! It is clear that opinions will differ on how wide such a range may be. The author in his preface states that "there is a need for a text-book devoted to this much-neglected branch of surgery". The reviewer feels that it may be a pity to divide surgery in such a manner. Is it felt further that surgeons may divide into those who operate only on the ambulatory and those who specialize in the bedfast patient? Surely those who understand the principles of surgery must also appreciate the problem which ambulation provides and which the author dismisses in half a page of a 900-page book. Is, perhaps, Minor Surgery or General Practitioner Surgery a better title? Yet this can hardly be so when consideration is given to the subjects discussed. Elevation of depressed fracture of the zygoma, adult hare-lip (facial carbuncle and cellulitis included, though even the author agrees that rest is here desirable), dentigerous cysts and ameloblastomas, carcinoma of the lip and tongue, acute and tuberculous cervical lymphadenitis, sacrococcygeal cysts, hæmorrhoids, acute suppurative tenosynovitis, dislocated knee-joints, are but a few picked at random. In fairness to the author, at the close of many sections he points out that the condition demands hospital care, i.e., bed—in such a case, surely, the subject is then irrelevant to the book title. It is, perhaps, proper at this stage to point out that the surgical methods described by Dr. Ferguson and his associates are in the main up to date, accurate, informative, and well presented. The author does not restrict himself to operative measures, but also deals with pathology and clinical appearances. It is doubtful whether these long descriptive sections add to the appeal of the book; they might be more properly left to the general text-books and the authors concentrate on ambulatory treatment exclusively, and only including clinical manifestations where these are necessary to prevent confusion in the selection of the appropriate ambulatory method. Thus, if the book is intended to be an "office book of reference in treatment for the ordinary practitioner", it seems unnecessary to include pages on aetiology and pathology of primary thrombosis of the axillary veins; or on aetiology, pathology, symptoms, and diagnosis of tennis elbow, to name but two. A section on human bites, with definition (surely the term is self-explanatory), a discussion on rabies, and a section

" description and habitat of the black-widow spider ", indicate that the author's range is wide and that he leaves the strict paths of surgery proper for the main thoroughfares of medical knowledge, albeit always delightfully and probably accurately. The author leaves nothing to the reader's imagination; thus *Fig.* 246, which shows " requirements for anal examination ", depicts: (1) a human eye, (2) a finger, (3) finger cot, (4) an anus, all drawn separately. Surely, at least, No. 4 could be taken for granted! The author also includes a diagram of the doctor's office and picture of its contents. *Fig.* 12, showing an electric stove and double boiling saucepan, again indicates how little is left to the reader, except to believe. The book is produced by J. B. Lippincott Co., and, therefore one need hardly say, is beautifully easy to read, with clear photographs, diagrams, and drawings.

Pye's Surgical Handicraft. Edited by HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London, etc. Thirteenth edition, fully revised. 8⅝ in. × 5½ in. Pp. 536 + xi, with 534 illustrations, some coloured. 1942. Bristol: John Wright & Sons. 25s. net.

THIS work first appeared in 1884, and this is the third edition under the present editorship since 1938.

We may confidently predict a long life of usefulness for it, since it has already survived the destruction of the plant and illustrations by two distinct acts of enemy bombing; once in the Bristol offices, and once in the new offices at Weston-super-Mare. Forty writers have collaborated with the editor in the present edition. Many of the subjects of which it treats would have been a novelty to the original writer, Mr. Pye, such as the chapters on pathology, X rays, blood transfusion, the use of sulphonamide, medico-legal problems, and those of compensation. Both author and publishers are to be warmly congratulated on the pluck and perseverance which the production of this work have entailed.

Surgery of Modern Warfare. Edited by HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London; etc. Second edition (complete in two volumes). 9¾ × 6⅝ in. Volume II. Compiled by 30 contributors. Pp. 481–1000 + xii, with 544 illustrations, many in colour. 1942. Edinburgh: E. & S. Livingstone. 40s. net.

THE second volume of this work is filled with practical matter. The following are among the most important of its chapters: Burns and frost-bite, by Surgeon Rear-Admiral Wakeley. This supplies a most welcome summary of what has become a rather complicated subject. The special dangers of tannic acid, with its liability to produce cicatrization, the use of gentian violet, and the application of the Bunyan-Stannard bag are all clearly described. The technique of the administration of chemotherapy is described by Dr. Thrower; the injury of peripheral nerves, by Professor Seddon; the methods of application of plaster-of-Paris, the applying of extension to limbs, and the use of the Thomas's splint and the Braun splint; wounds of the bones, by Robert Milne and Hamilton Bailey; wounds of joints, by Irwin and Surgeon Rear-Admiral Sir W. de Courcy Wheeler; amputations, by Rear-Admiral Sir W. de Courcy Wheeler and Jenner Verrall. The section on the ear is by Mr. Negus; wounds of the eye and orbit by Major H. B. Stallard; and perforating wounds of the eyeball by Mr. Charles Goulden and Mr. M. H. Whiting.

War Injuries of the Chest. Edited by H. MORRISTON DAVIES and ROBERT COOPE. 7¼ × 5 in. Pp. 131 + viii, with 36 illustrations. 1942. Edinburgh: E. & S. Livingstone. 6s. net.

THIS small handbook attempts to fill the gap between the articles on this subject scattered throughout the medical journals and large works on the surgery of the chest. The attempt achieves some measure of success. It is difficult to escape the impression that these two well-known thoracic experts are writing on one branch of their speciality in which their actual clinical experience has not been large; this lack of clinical material has probably been felt in all the E.M.S. Chest Units as a whole, and the general surgeon must still wait for an authoritative and instructive analysis on an impressive series of war wounds of the chest. The surgeon inexperienced in thoracic work will not find here clear indications for the employment of major operative work: it is probable that the authors in fact have not carried out many major interventions: this criticism should not be taken to imply that they should have done so! There is indeed a feeling amongst surgeons in this country that the immediate mortality-rate after formal thoracotomy in air-raid casualties has been high; such an impression can only be confirmed or dispelled by the publication of an adequate series of cases from the Chest Centres.

That all hæmothoraces should be completely aspirated is the opinion of thoracic physicians and surgeons in this country, and the reasons and methods for this are fully explained. It is impossible, however, not to criticize the advocacy of a general anæsthetic for the first aspiration—this is surely unwise and unnecessary. Nor will their opinion that a sterile hæmothorax should be washed out with saline gain much support. The instructions on the management of the infected hæmothorax are not sufficiently clear, nor full enough. There is no mention of the value of substituting rib resection and closed drainage for an intercostal catheter when the latter is repeatedly blocked: they prefer to proceed to open drainage.

In the chapter on anæsthesia, nitrous oxide is condemned as the most dangerous anæsthetic. Most thoracic units will find this hard to believe.

The arrangement of the book is good, and the chapters on anatomy, physiology, and pathology are excellent, though few will agree with their nomenclature of the broncho-pulmonary segments. The convenient size of the book—it will sit comfortably in a great-coat pocket—will make it attractive to many. In spite of the defects, it should be a most useful publication, and of the greatest value to those who do not understand the physiological mechanics of the chest.

Aids to Surgical Anatomy. By J. S. BAXTER, M.B., M.Sc., F.R.C.S.I., Lecturer on Anatomy, Bristol University; etc. Second edition. 6⅜ × 4 in. Pp. 193 + viii, with 26 illustrations. 1942. London: Baillière, Tindall & Cox. 4s. 6d. net.

THE first edition of this member of the now well-known " Aids " series appeared in 1931, and was written by Dr. R. H. Hunter, who has now retired from active teaching. The work of revision for the second edition has been carried out by Dr. Baxter, of Bristol. We have little quarrel with the anatomical aspect of this little book, though for a moment we wondered what the pharyngo-tympanic tube was (pp. 47 and 53). Do otologists really pass pharyngo-tympanic instead of Eustachian catheters these days? The surgical aspects are not as modern as they might

be. Amputations through the knee, the lower third of the leg, and at the mid-tarsal joint are so rarely practised to-day that reference to them in a small work such as this might well be omitted, whereas the standard procedures should be emphasized and amplified. The Hartel route for injecting the Gasserian ganglion is not mentioned, yet it has almost entirely replaced the trunk injection through the sigmoid notch of the mandible referred to on p. 25. We feel we must not be too hard on Dr. Baxter for his use of the Birmingham nomenclature, but it does seem a little inconsistent to mention Shrapnell's membrane, and yet refer to what is commonly known, anyhow in the British-speaking world, as Macewen's triangle as merely the supramental triangle without any mention of Sir William Macewen. Why call the submaxillary gland (p. 39) the submandibular gland, since nine out of ten, possibly, ninety-nine out of a hundred, practitioners to-day call this salivary gland the submaxillary gland?

To our way of thinking the only anatomical nomenclature which should, and will, survive in the long run, is that in current and constant use by surgeons and practitioners and, perhaps to a lesser extent, by anatomists themselves. Ultimately, usage alone determines the adoption and survival of a particular term, i.e., how valuable it proves to be in practice, and this criterion would appear to apply equally well to scientific terms as to language generally. We should, therefore, like to see more frequent use of the current anatomical terms than any rigid attempt to follow some system of terminology such as the so-called Birmingham and Basle nomenclatures.

There is much of value in this little book for the student of surgery, and we hope for an early appearance of a third and more modern edition. Dare we hope also for more widespread use of the everyday terms of anatomical parlance?

Illustrations of Regional Anatomy. By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. 8 × 6¼ in. In seven parts. Section I, Central Nervous System, fourth edition, 50 plates, 8s. 6d. Section II, Head and Neck, fourth edition, 64 plates, 15s. Section III, Abdomen, fourth edition, 43 plates, 7s. 6d. Section IV, Pelvis, fourth edition, 34 plates, 5s. Section V, Thorax, fourth edition, 33 plates, 5s. Section VI, Upper Limb, third edition, 42 plates, 8s. 6d. Section VII, Lower Limb, third edition, 52 plates, 10s. 6d. 1942. Edinburgh: E. & S. Livingstone. Bound volume, 60s. net.

THE value of such atlases (or, more correctly, collections of diagrams) as the present, has elicited much difference of opinion, although, if they are used as intended, namely, as adjuncts to lecture notes and alongside actual dissections, and not employed to replace a thorough practical acquaintance with the 'geography' of the body, they can be considered to serve a useful purpose. The author's original blackboard illustrations on which they are based were undoubtedly valuable, in that they were *gradually built up* in class alongside the demonstration of actual dissections, and thus served the purpose of stressing important topographical relationships. Whether the *completed* illustrations are as valuable is doubtful. Their chief merit, at least as regards some of the illustrations, lies in their simplification of 'difficult' parts. With the modern tendency to cut down the time in the curriculum devoted to dissection, the value of this production will become enhanced, but it will not constitute a royal road to the learning of anatomy. The whole work is very attractively produced and reasonably priced. Strongly contrasting colours are employed so that individual details can be readily discerned, and great care has been exercised over the insertion of the leader lines. The diagrams have a high standard of accuracy, though many are somewhat simplified representations of the actual conditions prevailing in the body. The present-day anatomist is concerned largely with the correlation of form and function, and the present work can only be recommended as an aid to visual memory on matters of pure topographical interest. They are particularly valuable to junior demonstrators of anatomy making their first acquaintance with the art of blackboard illustration, and to some extent the practitioner might find them of use to refresh his memory.

Gray's Anatomy. Descriptive and Applied. Edited by T. B. JOHNSTON, M.D., Professor of Anatomy in Guy's Hospital Medical School, University of London; and J. WHILLIS, M.D., M.S., Reader in Anatomy, Guy's Hospital Medical School, University of London. Twenty-eighth edition. 9¾ × 6 in. Pp. 1558 + xx, with 1347 illustrations, 631 in colour. 1942. London: Longmans, Green & Co. 60s. net.

ALTHOUGH it is only some four years since the last edition of this well-known book on anatomy was published, yet this has been necessary because the former edition has been exhausted.

This new edition shows signs of active revision and improvement, especially those chapters dealing with ductless glands, neurology, and the digestive system. The volume embraces both descriptive and applied anatomy, and can be looked upon as the best book in the English language on this subject.

The introduction of some new radiographs will be welcomed by the student, especially now that they are used in practically every anatomy examination both in anatomy and surgery, and the teaching of anatomy cannot be divorced from the latter subject.

Some new diagrams and drawings have appeared from the able brush and pen of Mr. A. K. Maxwell and thoroughly enhance the value of the book.

Few medical men can afford to be without this volume, which is absolutely comprehensive, and is *the* book for reference.

The Principles of Neurological Surgery. By LOYAL DAVIS, M.S., M.D., Ph.D., D.Sc. (Hon.), Professor of Surgery and Chairman of the Division of Surgery, Northwestern University Medical School, Chicago, Illinois. Second edition, thoroughly revised. 9 × 5⅞ in. Pp. 503, with 298 illustrations and 5 coloured plates. 1942. London: Henry Kimpton. 35s. net.

THE second edition of this book has been completely revised and brought up to date. It covers the whole ground of modern neurological surgery in a very comprehensive way. The illustrations are excellent and are beautifully reproduced, especially the microphotographs.

This book will probably teach the young neurosurgeon more than any other volume. It is not overcrowded with minutiæ, but is written in a definite dogmatic lucid style.

We can thoroughly recommend the book.

Encephalography. By E. GRAEME ROBERTSON, M.D. (Melb.), M.R.C.P. (Lond.), F.R.A.C.P., Physician to Out-patients, Royal Melbourne Hospital; etc. 9¾ × 7¼ in. Pp. 105 + ix, with 68 illustrations. 1942. Melbourne and London: Macmillan & Co. Ltd. 32s. 6d. net.

THIS monograph is the second to be published from the Walter and Eliza Hall Institute of Research in Melbourne.

There can be no doubt that monographs are the right and proper medium through which specialized knowledge can be disseminated.

The present volume is devoted to a very specialized technique used by the author. Nowadays encephalography is gradually replacing the older method of ventriculography, and it has the great advantage that no actual operation is necessary and the mortality is extremely low. In the author's series of two hundred and eight cases, one patient died thirty-six hours after the investigation.

There are some sixty-eight illustrations in this book, the majority being radiographs depicting various encephalograms, some of which are normal, while others illustrate pathological conditions.

This monograph will take its place as one of the books of real value, not only to neurologists and neuro-surgeons, but also to anatomists. It will long remain as the standard reference volume on encephalography.

A Study of the Blood in Cancer. With Special Reference to the Needs of the Tumour Clinic. By O. CAMERON GRUNER, M.D. (Lond.). 10¼ × 6⅝ in. Pp. 100 + xii, with numerous illustrations, some coloured. 1942. Montreal: McGill University (London: H. K. Lewis & Co.). 15s. net.

THIS book describes the technique for carrying out an elaborate analysis of the elements of the blood which, it is claimed, gives reliable information in from 83 to 97 per cent of cases of cancer. The results have not been submitted to statistical analysis, and their accuracy demands very considerable experience on the part of the observer. It is abundantly clear that the tests proposed could not be employed in the routine hospital investigation of patients by pathologists or technicians trained in orthodox hæmatological methods. It is disappointing to find that all the tests have an empirical basis and throw no light on the aetiology of the malignant process.

Non-pulmonary Tuberculosis. By MICHAEL C. WILKINSON, M.B., B.S., M.R.C.S., L.R.C.P., Medical Superintendent, Essex County Council Hospital, Black Notley. With a Foreword by Sir HENRY GAUVAIN, M.D., M.Chir., F.R.C.S. 7¼ × 4¾ in. Pp. 174 + xvi, with 12 plates. 1942. London: Hamish Hamilton Medical Books. 10s. 6d. net.

As Dr. Wilkinson's appointments show, he is well qualified to write on this subject: his book is based on the investigation and treatment of 593 patients during 1930–7 and on a detailed follow-up of most of these. For this alone the work deserves high commendation; so often a specialist who sees large numbers of a particular type of case makes no effort to collect his material, both for his own benefit and that of others.

This book, which is small and very readable, contains an account of non-pulmonary tuberculosis together with prognosis, which should be of interest to all and of great value to many. Students and those working for the higher examinations in surgery should not fail to read it. A criticism that can be made is that at times the pathology is of doubtful soundness. The statement made on page 53 that it is rare for tuberculosis of tracheobronchial glands to be associated with hæmatogenous lesions, and in this resembles tuberculosis of other lymphatic glands, would not be generally accepted, and, indeed, is contradicted on page 118, when the association of osseous lesions with tracheobronchial tuberculosis is admitted.

A particularly fortunate atmosphere is engendered in the book by insistence on the essential need for proper general treatment for all patients suffering from any tuberculous lesion; it is still only too common for the sufferer from an accessible and excisable tuberculous lesion (such as kidney or testis) to have the organ promptly removed and nothing else done.

Traumatic Surgery of the Jaws. Including First-aid Treatment. By KURT H. THOMA, D.M.D., Professor of Oral Surgery, and Brackett Professor of Oral Pathology, Harvard University. 10¼ × 6¾ in. Pp. 315, with 282 illustrations. 1942. London: Henry Kimpton. 30s. net.

THIS book has appeared at a time when both surgeons and dentists are looking for practical methods of treating every kind of injury to the jaws. Total warfare and the black-out have caused a spate of jaw injuries. Professor Thoma has written a book which will prove of real value to the dental surgeon of to-day in the first aid and emergency treatment of jaw injuries. It will also be read with advantage by the medical officer in the Services, for it contains just that amount of practical detail that is of real value.

Perhaps the treatment of fractures of the lower jaw by skeletal fixation will interest the general surgeon most of all, as this method has already had extensive trial in this country and has proved its worth.

The illustrations in this book are numerous and beautifully reproduced; they greatly enhance the value of the volume.

Synopsis of Ano-rectal Diseases. By LOUIS J. HIRSCHMAN, M.D., F.A.C.S., Ex-Vice-President, A.M.A.; Professor of Proctology, Wayne University; etc. Second edition. 7⅞ × 4⅞ in. Pp. 315, with 182 illustrations and 12 colour plates. 1942. London: Henry Kimpton. 22s. 6d. net.

THIS book is a synopsis of the author's larger and well-known text-book, *Diseases of the Rectum*, and in arrangement it follows the same lines. It is designed primarily for post-graduates, but also for general practitioners and students, and is of a convenient size for the pocket.

Nearly half of the book is devoted to anatomy, methods of examination and anæsthesia, and chapters on constipation and pruritus ani. These are well set out and give the student of proctology sound and practical advice. Chapter II on "Symptoms which should call attention to the rectum" is clear and helpful. Later sections on fissure and fistula do not so well accord with present-day practice, perhaps largely owing to the author not recognizing Peter Thompson's description of the sphincter ani externus, or Milligan and Morgan's classification of fistula-in-ano. Chapter X on abscess of the ano-rectal region leaves a good deal unsaid, and the management of deep rectorectal and pelvirectal abscesses is not entered into. As in most American books on the

rectum, much emphasis is laid on anal papillæ and anal crypts and cryptitis, and treatment of these conditions under local anæsthesia is clearly described. In connexion with hæmorrhoidectomy and excision of anal hæmatomas we are glad to see that the author discountenances the use of sutures for the skin wounds and recommends that good external drainage should be established by means of radiating incisions extending out on to the peri-anal skin.

The reader will see that although certain major conditions such as carcinoma of the rectum and complete prolapse are mentioned and their diagnosis discussed, treatment is not described. Nevertheless, this small book is only a synopsis; we cannot fail to admire the amount of information which Dr. Hirschman has been able to compress within such a small volume, and it well maintains the author's long and distinguished record in proctology. The book is profusely illustrated and has some excellent coloured plates and sigmoidoscopic drawings.

Minor Surgery (Oxford Medical Outline Series). By CHARLES M. OMAN, M.D., F.A.C.S., Rear-Admiral, Medical Corps, U.S. Navy, etc. $8\frac{3}{8} \times 5\frac{1}{2}$ in. Pp. 165 + viii, with interleaves. 1942. London: Oxford University Press. 12s. 6d. net.

IN this little book the salient points of minor surgery are outlined in tabulated form, under such headings as Definition, Classification, Treatment, Methods, General Remarks, etc. Blank pages are interleaved with those constituting the text and are, no doubt, for its amplification, and for notes of cases and other comments of the user. The author tells us that this outline has no pretence to text-book standing, but is aimed to serve as a practical working guide, and as such we can recommend it to the student, particularly when he is at work in the casualty and out-patient departments of his hospital. The fact that there are no illustrations suggests a further use still for the blank pages, at the hands of the student who is able to make pencil sketches. Just what constitutes minor surgery is perhaps as controversial a question as just what constitutes orthopædics, but Admiral Oman anticipates any criticism along these lines by pointing out that it must never be forgotten that all minor surgery is potentially major surgery. References to compound fractures, abdominal injuries, and osteomyelitis, therefore, which without any doubt constitute major surgery, are, no doubt, admissible in this respect. In terse, tabulated form the subject is covered well, and we can recommend this little book as a working notebook.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Surgical Diseases of the Spinal Cord, Membranes, and Nerve Roots. Symptoms, Diagnosis, and Treatment. By CHARLES A. ELSBERG, M.D. With chapters by CORNELIUS G. DYKE, M.D., and ABNER WOLF, M.D. 10 × 7 in. Pp. 598 + x, with 249 illustrations. 1942. London: H. K. Lewis & Co. Ltd. £4 net.

Illustrations of Surgical Treatment. Instruments and Appliances. By ERIC L. FARQUHARSON, M.D., F.R.C.S.E., Major, R.A.M.C., late Tutor in Clinical Surgery, Royal Infirmary of Edinburgh. Second edition. $9\frac{1}{2} \times 6$ in. Pp. 392 + xii, with 317 illustrations and 63 plates. 1942. Edinburgh: E. & S. Livingstone. 25s. net.

Nasal Sinuses. An Anatomical and Clinical Consideration. By O. E. VAN ALYEA, M.D., Assistant Professor, Department of Laryngology, Rhinology, and Otology, University of Illinois College of Medicine, Chicago. 9 × 6 in. Pp. 262 + xii, with 82 illustrations, some coloured. 1942. Baltimore: The Williams and Wilkins Co. (London: Baillière, Tindall & Cox.) 36s. net.

A Textbook of Gynæcological Surgery. By Sir COMYNS BERKELEY, M.A., M.C., M.D. (Cantab.), F.R.C.P., F.R.C.S., M.M.S.A. (Hon.), F.R.C.O.G., Consulting Gynæcological and Obstetric Surgeon to the Middlesex Hospital, etc.; and VICTOR BONNEY, M.S., M.D., B.Sc. (Lond.), F.R.C.S., F.R.A.C.S., M.R.C.P., Consulting Gynæcological and Obstetric Surgeon to the Middlesex Hospital, etc. Fourth edition. $9\frac{3}{4} \times 6\frac{1}{8}$ in. Pp. 912 + xii, with 574 original drawings by Victor Bonney, and 17 colour plates. 1942. London: Cassell & Co. Ltd. 50s. net.

Textbook of Surgical Treatment. Including Operative Surgery. Edited by C. F. W. ILLINGWORTH, M.D., Ch.M., F.R.C.S.E., Regius Professor of Surgery, University of Glasgow. Compiled by Eighteen Contributors. $9\frac{3}{4} \times 6\frac{1}{8}$ in. Pp. 528 + xii, with 144 illustrations. 1942. Edinburgh: E. & S. Livingstone. 28s. net.

Surgical Care. A Handbook of Pre- and Post-operative Treatment. By R. W. RAVEN, F.R.C.S., Major, R.A.M.C., late Surgeon, E.M.S.; Assistant Surgeon, Royal Cancer Hospital; etc. $7\frac{1}{4} \times 4\frac{7}{8}$ in. Pp. 271 + viii, with 80 illustrations. 1942. London: Edward Arnold & Co. 10s. 6d. net.

The Surgery of Pancreatic Tumours.—By ALEXANDER BRUNSCHWIG, M.S., M.D., F.A.C.S., Professor of Surgery, University of Chicago. Illustrated by GLADYS MCHUGH. 10 × $6\frac{7}{8}$ in. Pp. 421, with 124 illustrations. 1942. London: Henry Kimpton. 37s. 6d.

Recent Advances in Anæsthesia and Analgesia (including Oxygen Therapy). By C. LANGTON HEWER, M.B., B.S. (Lond.), D.A. (R.C.P. and S. Eng.), Senior Anæsthetist, St. Bartholomew's Hospital, etc. Fourth edition. 8 × $5\frac{1}{4}$ in. Pp. 341, with 135 illustrations. London: J. & A. Churchill Ltd. 18s. net.